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THE ACTION OF ELECTRICALLY CHARGED COPPER UPON CERTAIN ORGANISMS IN WATER.

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That metallic copper, when placed in water containing algæ or certain bacteria, will kill these organisms seems to be beyond doubt. That the area of copper surface exposed must be quite extensive and that the period of contact is of considerable duration is shown, also, in the experiments already published by a number of investigators. The formation of colloidal copper is, under natural conditions, slow, hence the tardiness in germicidal activity. Could this production of colloidal metal be hastened it is quite possible that a marked reduction in the lethal time would ensue. For the preparation of such solutions the electric current has been found most satisfactory, hence a trial has been made of the action of charged copper plates on certain pathogenic organisms suspended in river water.

As a preliminary experiment, copper foil was placed in a measured quantity of water for twenty-four hours, after which it was removed and weighed. The loss in weight for the same sample of foil was quite constant, being about 0.0006 gram for six square inches of copper surface in twenty-four hours. These copper strips when serving as anode and cathode, with a current of 4 volts and less than 0.01 ampere, lost copper at the rate of 0.0039 gram in one hour. This determination was made a number of times with closely agreeing results.

Other samples of copper-foil gave, under similar conditions, results agreeing among themselves but differing from those already

cited. One sample of copper was, therefore, used throughout this work.

Having determined the rate of removal of the copper from the electrodes, they were placed in water containing B. typhosus. In another experiment B. coli was used. The water was from the laboratory tap, and was sterilized in an autoclave. Seeding was from an aqueous suspension of the organisms grown on agar slants for eighteen to twenty-four hours at incubator temperature. Control experiments made with water and organisms alone showed practically no change at the end of the experiment period. To determine the action of the current without the copper, platinum strips were substituted, with the result shown in Table 1, *C*.

TABLE I.—Electrode surface 6 square inches. Current 4 volts, and less than 0.01 ampere. Volume 1000 c.c., 1 c.c. plated.

A.

	Before treating.	15 minutes, copper 1 to a million.	30 minutes, copper 2 to a million.	45 minutes, copper 3 to a million.
B. typhosus	40,000	115	60	1
	44,000	230	20	1
	107,520	80,500	60	1
	100,400	76,420	36	0

B.

	Before treating.	15 minutes, copper 1 to a million.	30 minutes, copper 2 to a million.	45 minutes, copper 3 to a million.
B. coli communis	Innumerable.	Innumerable.	89,000	55,600
			80,000	54,500

C.

	Before treating.	15 minutes, platinum electrodes.	30 minutes, platinum electrodes.	45 minutes, platinum electrodes.
B. typhosus	34,000	24,800	16,200	8,640
	30,000	21,600	18,900	

Is it possible to pass water at the intake of a reservoir over charged copper plates at such a rate that pathogenic organisms, particularly B. typhosus, are destroyed in transit? If not killed in transit, would the copper taken up by the water be sufficient to kill them within a reasonable time?

The following very primitive device was used to simulate a reservoir with intake and outlet.

A glass jar of four litres' capacity was fitted with two perforated copper disks, forming the positive and negative poles and exposing about 31 square inches of surface. A wooden cover, carrying inlet and outlet tubes as well as the wires for the electric supply, fitted the top of the jar accurately. Its joint was packed with cotton-

wool as were also the openings for wires and tubes. The entire piece of apparatus, except the copper disks, was sterilized in an autoclave. The copper was sterilized by igniting several times in alcohol, thereby preserving its brightness. A supply jar at a higher level carried an infected stream of water to the bottom of the reservoir whence it flowed up, over and through the perforated disks, and out by a siphon tube, dipping just below the surface of the liquid. Screw clips regulated the rate of flow, and every care was taken to guard against contamination. The amount of copper lost was found by weighing the disks, and was 0.00032 gram per minute. After five minutes' action, then, a litre of water contained 1.6 part copper per million. Plates were made immediately upon the exit of the effluent, and again after it had stood at room temperature for one hour. Controls in water, alone, showed practically no change at the end of thirty minutes nor after one hour.

TABLE II.

	Before treating.	1 minute.	2 minutes.	5 minutes.
B. typhosus	102,600	1360	1380	729
	98,600	1870	1300	900
	180,000	1700	.....	1090
	170,000	1800	.....	1020

	Before treating.	1 min.	2 min.	5 min.	10 min.
B. coli communis	42,120	3300	2400	980	485
	42,660	4116	2520	1060	600
	43,200	.....	2400	1392	864
	45,900	.....	2320	1104	605

After standing for one hour plating the effluent showed the number of organisms per cubic centimetre to be as follows:

Time.	B. typhosus.	B. coli communis.
1 minute	36	22
2 minutes	7	2
5 "	0	0
10 "	0	0

A comparison of the foregoing experiments would seem to indicate a balance in favor of a large copper surface with which the organisms come in contact, rather than the actual copper content of the water. Accordingly, another apparatus was arranged in which the water passed over four plates, placed about an inch and a half apart in a glass tube holding 200 c.c.

The copper disks, perforated as before, gave a surface of 9.6 square inches. As the disks were four in number the organisms in their descent were compelled to pass over the four separate surfaces, an interval elapsing between leaving one and reaching the next. Electrolyzed in tap-water, they showed a loss of 0.00014 gram copper per minute for 4 volts and 0.02 ampere. Portions of

500 c.c. were used, giving the water a copper content of 1.4 parts per million for five minutes and 2.8 parts for ten minutes.

TABLE III.

	Before treating.	5 minute .	10 minutes.
B. typhosus .	,440	sterile.	sterile.
	50,200	"	"
	22,140	1	"
	28,900	3	"

In the making of these experiments the water in which the copper had been electrolyzed was subjected to the ferrocyanide test, and it was noted that when the organisms were not added to the water the reaction was much more distinct than that given by the same quantity of copper in the presence of the bacteria. In the latter case it was sometimes impossible to obtain any reaction with ferrocyanide.

SUMMARY. 1. Copper electrodes carrying a small current are actively germicidal for B. typhosus and B. coli communis.

2. B. typhosus is the more easily affected.

3. That it is the copper which plays the important role is shown by substituting for it platinum electrodes.

I am greatly indebted to Prof. Edgar F. Smith, of the John Harrison Laboratory of Chemistry, University of Pennsylvania, for placing at my disposal the facilities of the electrochemical laboratory, wherein the electric part of this work was carried out.

STUDIES ON THE BACTERICIDAL ACTION OF COPPER ON ORGANISMS IN WATER.

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THE investigations of Moore and Kellerman[1] on the employment of copper for the destruction of algæ, and incidentally of bacteria, in water, have developed an excellent method for the destruction of certain algæ which have caused a great deal of annoyance, the presence of these cryptogams in water frequently causing a disagreeable odor and taste rendering it unfit for household, and, in many instances, industrial purposes. Such waters while they are not, in the present state of our knowledge on the subject, looked upon as being especially detrimental to the health of a community, will be condemned, and justly, on account of their disagreeable

[1] Bulletin, No. 64, Bureau of Plant Industry.

odor and taste: The same community, however, will use bacteriologically impure water, a much more dangerous water, without considering the question of impurity. The latter, being in many cases clear, odorless, and tasteless, does not appeal to the senses as do those containing algæ, and in spite of all that has been said and done, it is exceedingly difficult to prevail upon many of the populace to take measures which will remove a contamination that is not manifest, *i. e.*, cannot be detected by appearances, and therefore to them does not exist. Should these waters become turbid or show other evidences of pollution they are ready for any measure that will remove the objectionable features. Manifestly, the work of the above-mentioned investigators has been of great value from both a sanitary and an æsthetic standpoint. Furthermore it has caused a great deal of discussion and controversy over one of the most vital questions confronting the sanitarian—namely, that of the destruction of bacteria in contaminated waters. They have shown conclusively, both by laboratory experiments and the practical application of the method, that the repeated treatment of small bodies of water (lakes, ponds, reservoirs, etc.), at comparatively long intervals with small amounts of cupric sulphate will keep them free from objectionable algæ. When we approach the question of destroying bacteria in water by such methods certain factors must be taken into consideration, viz.—the contamination usually being continuous, or at frequent and irregular periods, the bactericidal substance must be used continuously and, for various reasons, in as small quantities as possible; therefore the question must be considered from the following standpoints:

(1) Are bacteria destroyed by very dilute solutions of copper salts or by copper in any form?

(2) Which of the copper salts is the most active, and at the same time cheap enough to prevent the question of expense being so great a factor as to exclude its use?

(3) What influence would organic and inorganic substances, from various sources, have on the action of the copper?

(4) Would the constant ingestion of small quantities of copper have a detrimental effect on the health of an individual using water so treated?

While the latter question will not be discussed in this paper we wish to state, for the benefit of the laity and many of the profession who entertain erroneous ideas regarding the subject, that it is not the object of sanitarians, who advocate the use of copper for the purification of water, to use it to the exclusion of other approved methods, such, for instance, as filtration, but to employ it to supplement such methods when the filters are for any reason inadequate; when they are in process of construction; or when filters cannot be immediately installed. And we sincerely hope that work along this line will not convey the idea to anyone that filtration is unnecessary. Also, that

the writer, who when the question was first taken up was strongly prejudiced against the employment of copper for this purpose, has been unable to find any authentic records of chronic poisoning.

The investigation reported in this communication was undertaken to determine to what extent various bacteria are destroyed by dilute solutions of copper salts; and also to show which of these salts has the greatest bactericidal action. Incidentally a few experiments were made with colloidal copper.

The experiments include the action of these substances on B. typhosus, B. coli communis, B. dysenteriæ, B. cloacæ, B. proteus vulgaris, B. prodigiosus, and the staphyloccus pyogenes aureus.

These organisms were selected for the following reasons: Some are pathogenic bacteria causing diseases which frequently follow the use of contaminated waters, and are those which we especially desire to destroy; others while non-pathogenic are frequently, one might say constantly, found in such waters; and the various organisms, possessing different degrees of resistance to the action of bactericidal substances, give us a fair indication of the action of the substances on the majority of non-spore-forming bacteria.

METHODS OF PROCEDURE. Flasks containing the various solutions were inoculated with either a small amount of a twenty-four-hour bouillon culture, or a large loopful of bacteria scraped from the surface of a twenty-four-hour agar culture; the organisms were thoroughly distributed throughout the solution and agar plates made immediately, to serve as controls, and one-half and one hour after inoculation, then at intervals of one hour for twelve hours, and if all bacteria were not destroyed within the twelve-hour limit, again at twenty-four hours. The plates were examined at the end of twenty-four hours, and, taking into consideration the facts that some of the organisms employed developed more slowly than others, and that while some might not be destroyed yet their functions would be so inhibited that they would develop slowly, they were counted at the end of forty-eight and again at seventy-two hours, which we consider would be ample time for the development of all bacteria. At least five experiments were made with each organism in the various solutions and dilutions.

EXPERIMENTS ON THE ACTION OF CUPRIC SALTS. In these experiments the sulphate, chloride, acetate and nitrate were employed in solutions of 1 : 250,000, 1 : 500,000, 1 : 1,000,000, 1 : 1,500,000, 1 : 2,000,000, made with both distilled and tap-water.

Experiments show the bactericidal properties of the sulphate to be greater than that of the other salts; a solution of 1: 250,000, as a rule, destroying the bacillus typhosus and bacillus coli communis in less than one hour; in solutions of 1: 500,000 and 1: 1,000,000 they were usually destroyed in two to three hours, while in solutions of 1: 2,000,000 there was rarely more than 80 to 90 per cent. destroyed in ten hours, and frequently a few would be capable of developing

after twenty-four hours' exposure. Some strains of B. coli communis appear to be slightly more resistant than B. typhosus. The resistance of B. dysenteriæ, B. cloacæ, B. proteus vulgaris, and staphylococcus aureus is greater than that of the B. typhosus and B. coli communis. A solution of 1: 250,000 would not destroy the B. prodigiosus in less than five to six hours, and a 1: 500,000 solution would frequently require ten hours for the destruction of this organism. If, however, the bacteria were washed in distilled water before inoculation of the flasks, the resistance was found to have been lessened, due in all probability to the fact that some of the slimy substance which surrounds the bacterial cell and protects it, to some extent, from the action of deterimental agencies, is removed.

The other cupric salts were found to be less active than the sulphate in the following order: chloride, acetate, nitrate.

The following table will serve to show the difference in the action of the various salts on the B. typhosus and B. coli communis, and of the cupric sulphate on the various bacteria:

Colloidal Solutions. These experiments were made in both distilled and tap-water as follows:

(1) Inoculation of 100 c.c. flasks of water containing pieces of copper-foil of various sizes.

(2) Inoculation of copper vessels of 500 c.c. capacities.

In the first series of experiments pieces of polished copper-foil giving an exposed surface of from 0.5 to 3 c.c. were placed in an Erlenmeyer flask containing 100 c.c. of water. Some were inoculated immediately, others containing pieces exposing a surface of 0.5 c.m. were placed aside and allowed to stand for ten days before inoculation. In those inoculated immediately after the copper had been placed in the flasks, there was always a reduction in the number of bacteria, but in no instance over 50 per cent. in eight hours, while those in which the foil remained for ten days before inoculation gave results as follows:

Bacillus typhosus, 90 per cent. destroyed in two hours, 98 per cent. in three hours, and all in four hours; bacillus coli, 50 per cent. destroyed in two hours, 85 per cent. in three hours, 90 per cent. in four hours, and all in five hours; bacillus dysenteriæ, 83 per cent. destroyed in two hours, 98 per cent. in three hours; in four hours but twenty-five bacteria developed, and all were destroyed in five hours; bacillus cloacæ, 20 per cent. were destroyed in two hours, all were not killed until they had been exposed to the action of the solution for ten hours; bacillus proteus vulgaris, 97 per cent. destroyed in twenty-four hours; staphylococcus pyogenes aureus, 66 per cent. in two hours, at the end of four hours twenty-five colonies developed; in five hours all had been destroyed. The bacillus prodigiosus was more resistant, being decreased but 50 per cent. in twenty-four hours.

TABLE SHOWING ACTION OF THE VARIOUS CUPRIC SALTS ON THE B. TYPHOSUS AND B. COLI COMMUNIS

Organism.	Salt.	Dilution.	Average number to c.c. in control.	Per cent. destroyed in hours.											
				1	2	3	4	5	6	7	8	9	10	11	12	24
B. typhosus.	Sulphate.	1 : 250,000	110,000													
		1 : 500,000	100,000	99	100											
		1 : 1,000,000	140,000	78												
		1 : 1,500,000	120,000	50	85	99	100									
		1 : 2,000,000	130,000	4	7	55	80	82	87	87	93	98	100			
	Chloride.	1 : 250,000	100,000	99	100											
		1 : 500,000	92,000	98	100											
		1 : 1,000,000	95,000	10	20	32	50	62	75	83	92	92	93	94	94	98
		1 : 1,500,000	103,000	8	14	20	23	40	59	62	64	64	65	65	66	85
		1 : 2,000,000	114,000	5	10	14	16	21	29	33	34	34	34	35	35	42
	Acetate.	1 : 250,000	122,000	80	95	99	100									
		1 : 500,000	113,000	66	66	68	70	73	80	89	94	96	100			
		1 : 1,000,000	117,500	7	19	22	30	38	42	42	45	46	46	48	62	95
		1 : 1,500,000	121,000	5	10	19	25	28	35	36	40	41	55	49	50	75
		1 : 2,000,000	108,000	2	2	4	9	11	15	22	30	33	33	34	34	65
	Nitrate.	1 : 250,000	85,000	21	33	40	50	58	71	80	90	93	98	99	100	
		1 : 500,000	82,000	11	20	38	61	60	70	78	84	84	85	85	87	99
		1 : 1,000,000	91,000	5	10	10	10	11	12	30	32	33	35	37	42	90
		1 : 1,500,000	98,000	0	3	9	12	14	18	20	20	23	26	37	29	80
		1 : 2,000,000	79,000	0	0	5	8	12	13	22	22	22	24	25	25	71
B. coli communis.	Sulphate.	1 : 250,000	105,000	100												
		1 : 500,000	120,000	99	100											
		1 : 1,000,000	99,000	80	99	100										
		1 : 1,500,000	150,000	87	95	99	100									
		1 : 2,000,000	110,000	8	10	14	27	50	85	90	92	93	93	95	98	
	Chloride.	1 : 200,000	160,000	89	99	100										
		1 : 500,000	140,000	58	68	95	97	99	99	99+	100					
		1 : 1,000,000	100,000	20	24	31	38	50	54	65	65	66	67	75	80	98
		1 : 1,500,000	124,000	10	10	13	15	21	29	32	34	35	38	44	55	89
		1 : 2,000,000	120,000	2	5	7	13	17	19	23	23	26	29	33	35	73
	Acetate.	1 : 250,000	120,000	78	95	99	100									
		1 : 500,000	130,000	22	38	54	77	81	87	89	96	98	99	99	100	
		1 : 1,000,000	136,600	5	8	11	15	22	36	38	48	57	52	55	62	90
		1 : 1,500,000	120,000	3	10	10	11	13	16	22	37	...	...	...	...	76
		1 : 2,000,000	124,000	0	2	6	8	9	11	11	15	18	23	26	28	68
	Nitrate.	1 : 250,000	100,000	13	22	30	30	35	45	51	60	71	75	85	94	100
		1 : 500,000	100,000	5	10	12	20	20	25	30	35	41	49	58	72	99
		1 : 1,000,000	104,000	7	10	10	11	12	15	20	30	32	40	51	60	90
		1 : 1,500,000	90,000	0	2	5	7	8	11	15	25	31	31	34	38	68
		1 : 2,000,000	92,000	0	0	3	5	5	7	9	12	15	19	25	30	61

When copper vessels of 500 c.c. capacity, which gave a surface of 266 square centimetres exposed to the water, were employed the action was more marked, the B. typhosus and B. coli communis being, as a rule, completely destroyed within three hours. The B. dysenteriæ, B. cloacæ, and staphylococcus aureus and B. proteus vulgaris are more resistant. The B. prodigiosus always showed a marked reduction in numbers, but was rarely completely destroyed in twenty-four hours. It is interesting to note that two cultures of B. prodigiosus, from different sources, employed, while being but slightly decreased in numbers in two to three hours, had almost complete inhibition of their pigment producing function, after exposure to the copper for this period, regaining it, however, after cultivation

for two or three generations on suitable media. The copper must be kept highly polished, as after it has become tarnished the bactericidal action is markedly inhibited.

Flasks containing one litre of a 1: 500,000 solution of cupric sulphate were inoculated with relatively large quantities of bacteria, plated and reinoculated at intervals of from two to six hours.

TABLE SHOWING THE ACTION OF CUPRIC SULPHATE ON VARIOUS BACTERIA.

Organism.	Dilution.	Aver'ge No. per c.c. in control.	Per cent. destroyed in hours.												
			1	2	3	4	5	6	7	8	9	10	11	12	24
B. typhosus	1 : 250,000	110,000	100												
	1 : 500,000	100,000	99	100											
	1 : 1,000,000	140,000	78	100											
	1 : 1,500,000	120,000	50	85	99	100									
	1 : 2,000,000	130,000	4	7	55	80	82	87	87	93	98	100			
B. coli communis	1 : 250,000	105,000	100												
	1 : 500,000	120,000	99	100											
	1 : 1,000,000	99,000	80	99	100										
	1 : 1.500,000	150,000	87	95	99	100									
	1 : 2,000,000	110,000	8	10	14	27	50	85	90	92	93	93	95	98	100
B. dysenteriæ	1 : 250.000	11,500	95	100											
	1 : 500,000	120,000	40	95	97	100									
	1 : 1,000,000	72,000	31	50	62	85	99	99+	100						
	1 : 1,500.000	12,400	20	30	40	42	60	70	75	81	85	87	90	91	100
	1 : 2,000,000	13,600	20	20	25	30	30	45	60	62	62	68	71	75	98
B. cloacæ	1 : 250,000	92,000	90	99	100										
	1 : 500.000	86,000	68	90	99	100									
	1 : 1,000,000	101,000	33	85	89	96	96	99	100						
	1 : 1.500,000	89,000	5	10	30	40	70	85	95	98	98	99	100		
	1 : 2,000,000	94,500	3	10	31	40	80	83	85	88	91	91	98	97	100
B. proteus vulgaris	1 : 250,000	115,000	93	100											
	1 : 500,000	106,000	65	92	98	100									
	1 : 1,000,000	111,000	28	51	82	90	95	98	100						
	1 : 1,500,000	99,000	10	18	27	42	69	90	93	98	99+	100			
	1 : 2,000,000	118,000	0	12	22	36	51	65	78	80	85	88	92	95	100
B. prodigiosus	1 : 250,000	102,000	95	99	99	99+	100								
	1 : 500,000	109,900	15	26	80	95	97	99	99+	100					
	1 : 1,000,000	110,000	9	11	16	33	33	45	47	61	63	69	...	...	93
	1 : 1,500,000	107,000	4	12	17	30	38	46	46	48	51	52	60	61	92
	1 : 2,000,000	98,000	0	1	8	8	10	11	15	30	38	41	42	50	88
Staphylococcus pyogenes aureus	1 : 250,000	92,000	99	100											
	1 : 500,000	88,000	50	70	90	100									
	1 : 1,000,000	86,000	15	30	30	70	78	85	97	99	99	100			
	1 : 1,500,000	100,000	8	22	28	95	58	69	85	93	95	99	100		
	1 : 2,000,000	95,000	1	8	10	30	50	64	79	82	85	98	99	99+	100

The results show that after a series of inoculations the bactericidal action of the solution gradually decreases.

The action was greater when distilled water was used for making the dilution than when tap-water was employed. Very little difference was found between the distilled and the filtered water used in the laboratory. The bacteria from agar cultures were more readily destroyed than those in bouillon cultures; this might be explained by the fact that a small amount of bouillon, 0.05 to 0.1 c.c., was added to the solution with the bacteria.

The question arises: Why is the bactericidal action of the sulphate greater than that of the other copper salts? Many of the metallic salts precipitate albuminous substances—*e.g.*, those of mercury and copper; in fact, salts of most of the heavy metals. These by coagulating the protoplasm will kill the organism. In the precipitation there is formed a compound of the metal with the albumin. One would naturally consider that the compound containing the highest percentage of the metallic element would possess the greatest bactericidal activity. Such, however, is not the case, and we are compelled to look farther for an explanation. Krönig and Paul,[1] in an investigation on the action of various chemicals on bacteria, showed that certain compounds varied, in their action, in proportion to the degree of electrolytic dissociation. Copper sulphate undergoes dissociation into its positive and negative ions more readily than the other cupric salts. The copper ion combines with certain constituents of the bacterial cell, either destroying it directly or rendering the cell wall impermeable so that nutritive substances cannot be absorbed. Colloidal copper in all probability acts in the same manner.

The results obtained seem to warrant the following conclusions:

(1) Dilute solutions of copper salts have a marked destructive action on many bacteria. Of these salts the sulphate is most active. This is probably due to the fact that it undergoes electrolytic dissociation more readily than the others.

(2) The amount of sulphate to be used in the water should be from one part in 250,000 to one part in 100,000, depending on the character of the water.

(3) Colloidal copper will quickly destroy certain bacteria; should copper vessels or plates be used to destroy bacteria in water they must be kept highly polished or the bactericidal properties will be greatly reduced.

(4) We have been unable to find evidence of copper, ingested in small quantities for long periods, having a deterimental action on the health of an individual.

A STUDY OF THE ACTION OF COLLOIDAL SOLUTIONS OF COPPER UPON BACILLUS TYPHOSUS.

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The excellent work done some months ago by the Bureau of Plant Industry, Department of Agriculture, in Washington, on the destruction of algæ and certain pathogenic bacteria by the solution

[1] Zeitschrift f. Hygiene, 1897, Bd. xxv.

of salts of copper and the colloidal solution of copper led us to undertake this work along similar lines.

The small numbers of algæ in the waters of the Schuylkill and the Delaware Rivers have not, up to the present time, caused any special contamination of the reservoirs or the filters of the Philadelphia water supply.

The suspected contamination, however, of our river water with typhoid and the numerous epidemics of typhoid fever in this community make every method or means brought forward to diminish the death-list from typhoid fever worthy of the greatest attention and of most careful investigation.

By colloidal solution of copper is meant the solution of metallic copper that takes place when a bright copper surface is brought into contact with water.

Different grades of copper do not all give off the same amount of colloidal copper, and on account of the different bacterial content, and because of the varying amount of vegetable and mineral matter in solution, typhoid bacilli and water organisms are not always acted upon in exactly the same time or in the same way by colloidal copper solutions.

The waters of the Delaware and the Schuylkill rivers show a great variation in number of bacteria per cubic centimetre in different seasons. This variation is from 200 germs per cubic centimetre to many millions.

For comparison and control, this experimental work was not carried on in test-tubes, as was done in previous experiments, but in copper and other metallic vessels of different sizes varying from one-fourth litre to eight litres in capacity.

The following experiments given in detail were made to determine, if possible, the action of metallic copper upon typhoid organisms and upon the ordinary river-water organisms of both the Schuylkill and Delaware Rivers.

The action of metals—tin and aluminium—and of glass upon the same organisms was determined under the same circumstances as the action of copper.

The vessels used in these tests were made with a lid for filling, and a small opening in the lid closed with a plug of cotton for the convenient testing of the contents.

Experiment to Determine the Life of Typhoid Organisms in Copper, Tin, and Glass Vessels.

Copper, tin, and glass vessels of various sizes were filled with tap-water (Schuylkill), and sterilized in an autoclave at 18 pounds' pressure for thirty minutes.

These vessels were inoculated with 1 c.c. per litre of suspension of typhoid organisms in water and thoroughly shaken.

Plates were made in one hour, three hours, and in twenty-four hours.

The water in the glass vessels was exposed freely to the light; this, no doubt, had a marked influence upon the destruction of the organisms.

The experiment was conducted at room temperature.

The suspension of typhoid germs used was made in sterile water, and contained 270,000,000 organisms per cubic centimetre. One cubic centimetre of this suspension was used to each litre of water.

Character and size of the vessels.	No. of sq. inch surfaces exposed.	Count within 1 hour.	Count in 3 hours.	Count in 24 hours.
8 litre copper	287	4,400	0	0
8 litre tin	287	32,480	29,973	3,650
8 litre glass	330	12,300	14	0
4 litre copper	193	9,900	0	0
4 litre tin	193	25,280	44,500	6,700
4 litre glass	184.5	24,680	3,280	200
2 litre copper	124	5,460	0	0
2 litre tin	124	24,910	28,960	8,400
2 litre glass	130	8,400	18	0
1 litre copper	79	4,053	0	0
1 litre tin	79	52,930	40,760	9,660
1 litre glass	80	14,130	10	10
⅛ litre copper	48	6,646	0	0
⅛ litre tin	48	20,000	48,000	141,200
⅛ litre glass	52	45,000	15,400	640
¼ litre copper	25	1,960	0	0
¼ litre tin	25	25,600	68,130	7,900
¼ litre glass	28	14,260	148,000	176,850

In this experiment the typhoid organisms were all killed within three hours in the copper vessels.

In the glass vessels there was considerable variation due, possibly, to the differences in glass, and the tin vessels showed also a great variation in their effect.

Experiment to Determine the Life of Typhoid Organisms in Filtered Water in Copper Vessels.

In this experiment 250 c.c. of filtered water, showing an absence of germs, was placed in a sterile copper vessel of 250 c.c. capacity and an exposed surface of 25 square inches, and was inoculated with 1 c.c. of a suspension of typhoid organisms which contained 150,000,000 germs per cubic centimetre.

The germs were thoroughly mixed in the water, and plates were made in fifteen minutes, one hour, two hours, three hours and twelve hours.

Time of count.	Colonies per c.c.
15 minutes	30,000
1 hour	3,800
2 hours	1,200
3 "	0
12 "	0

Experiment to Determine the Life of Typhoid Organisms in Water Sterilized in a Glass Vessel in an Autoclave and Transferred to a Copper Vessel which had been Sterilized by Direct Flame and then Inoculated.

500 c.c. of water was sterilized in a glass vessel in an autoclave under 15 pounds' pressure for one-half hour and was, after cooling, poured into a sterile copper vessel, having an exposed inner surface of 48 square inches and inoculated with 2 c.c. of a suspension of typhoid in water containing 150,000,000 germs per cubic centimetre.

Counts were made in fifteen minutes, one hour, two hours, three hours, and twelve hours.

Time of count.	Colonies per c.c.
15 minutes	43,000
1 hour	1,570
2 hours	1,400
3 "	0
12 "	0

Experiment to Determine the Life of Typhoid Organisms in Sterile Tap-water in a Copper Vessel which had been Cleansed with Lemon-juice and Salt, and Rinsed with Hot Water.

In this instance 2 c.c. of a watery suspension of typhoid bacilli containing 12,000,000 germs per cubic centimetre were added to a two-litre copper vessel filled with sterile tap-water and left at room temperature.

Counts were made immediately in one hour, two hours, three hours and twelve hours.

Time of count.	Colonies per c.c.
15 minutes	43,860
1 hour	78
2 hours	11 (?)
3 "	0
12 "	0

Experiment to Determine the Life of Typhoid Organisms in Raw Tap-water in a Copper Vessel.

A four-litre copper vessel having an exposed surface of 193 square inches was cleansed thoroughly with lemon-juice and salt, and then washed in hot water.

The vessel was filled with tap-water having 1030 organisms per cubic centimetre and was inoculated with 4 c.c. of a watery suspension of typhoid organisms which contained 12,000,000 germs per cubic centimetre, and allowed to stand at room temperature.

The counts were made immediately, in one hour, two hours, three hours, and twelve hours.

Time of count.	Colonies per c.c.
Immediately	15,090
1 hour	15,000
2 hours	15,200 few typhoid.
3 "	13,800 no "
12 "	0

The rapidly growing water organisms obscured the typhoid organisms so quickly that the counts were difficult to make, and the experiment was repeated.

This experiment was repeated in a two-litre copper vessel filled with ordinary tap-water having 150 water organisms per cubic centimetre.

The exposed copper surface was 124 square inches.

The water was inoculated with 2 c.c. of watery suspension of typhoid organisms containing 3,360,000,000 germs per cubic centimetre and allowed to stand at room temperature (75°), and counts were made every fifteen minutes for three and a half hours.

Time of count.	Typhoid germs, per c.c.	Water germs, per c.c.
Immediately	18,000	150
¼ hour	12,000	110
⅓ "	7,800	110
¾ "	4,200	120
1 "	2,400	125
1¼ hours	2,160	112
1⅓ "	1,440	115
1¾ "	340	108
2 "	360	100
2¼ "	120	104
2⅓ "	24	95
2¾ "	0	90
3 "	0	92
3¼ "	0	60
3⅓ "	0	60

Experiment to Determine the Life of Typhoid Organisms in Sterile Tap-water Contained in a Copper Vessel.

A 500 c.c. copper vessel having an exposed surface of 48 square inches was cleansed with lemon-juice and salt, and washed with hot water and filled with sterile tap-water.

The water was inoculated with 1,081,000,000 typhoid organisms. Counts were made immediately and every fifteen minutes for three and a half hours.

Time of count.	Typhoid germs, per c.c.	Water germs, per c.c.
Immediately	18,000	0
¼ hour	1,280	0
⅓ "	82	0
¾ "	68	0
1 "	0	0
1¼ hours	0	0
1⅓ "	0	0
1¾ "	0	0
2 "	0	0
2¼ "	0	0
2⅓ "	0	0
2¾ "	0	0
3 "	0	0
3¼ "	0	0
3⅓ "	0	0

Experiment to Determine the Amount of Colloidal Copper in Solution.

A one-litre copper vessel was filled with tap-water and allowed to stand for three hours at room temperature (75°).

The surface of copper exposed to the action of water was 79 square inches.

A test was made by Dr. Robinson to find the amount of colloidal copper in solution.

He reported that there was one part of copper to 4,000,000 parts of water, or one square inch gave off one part to 316,000,000 parts of water.

In many of the experiments much less time than three hours was required to kill off all the typhoid organisms.

The action of infinitesimal amounts of copper is remarkable.

Experiment to Determine the Resistance of Typhoid Organisms and Schuylkill River Organisms to Colloidal Solutions of Copper.

One litre of Schuylkill water taken at Market Street was placed in a clean copper vessel.

A bacterial count made immediately showed approximately 400,000 germs per cubic centimetre.

Another litre of water from the same source was placed in a sterile glass beaker and kept in the dark.

A count of the bacteria in this water also showed approximately 400,000 organisms per cubic centimetre, counts of the water bacteria in the glass beaker every fifteen minutes for six hours were as follows:

Time of count.	No. of colonies.	Time of count.	No. of colonies.
¼ hour	400,000	3¼ hours	550,000
½ "	454,000	3½ "	530,000
¾ "	475,000	3¾ "	550,000
1 "	450,000	4 "	595,000
1¼ hours	450,000	4¼ "	560,000
1½ "	460,000	4½ "	550,000
1¾ "	450,000	4¾ "	560,000
2 "	475,000	5 "	580,000
2¼ "	480,000	5¼ "	560,000
2½ "	480,000	5½ "	560,000
2¾ "	480,000	5¾ "	560,000
3 "	500.000	6 "	600,000

To the copper vessel filled with Schuylkill water was added 3 c.c. of a suspension of typhoid organism containing about 2,888,000,000 germs per cubic centimetre.

Counts were made every fifteen minutes for six hours.

In one and three-fourth hours no typhoid organisms could be found in the water, that would grow on plates.

After three hours the water in the copper vessel was poured into a glass vessel and allowed to remain there, protected from the light.

One set of plates was allowed to remain at room temperature and another duplicate set was kept in an incubator at (37° C.).

In six hours the number of organisms remained stationary.

Time of count.	No. organisms per c.c. agar plates grown in incubator.	No. organisms per c.c. agar plates grown in room temp. 75°.
At once	960,000	960,000
¼ hour	950,000	920,000
⅛ "	925,000	923,000
¾ "	605,000	600,000
1 "	525,000	350,000
1¼ hours	350,000	168,000
1⅛ "	250,000 no typhoid.	120,000 no typhoid.
1¾ "	237,000 "	115,000 "
2 "	237,000 "	115,000 "
2¼ "	212,000 "	120,000 "
2⅛ "	187,000 "	95,000 "
2¾ "	180,000 "	95,000 "
3 "	175,000 "	95,000 "
3¼ "	168,000 "	90,000 "
3⅛ "	150,000 "	75,000 "
3¾ "	120,000 "	50,000 "
4 "	105,000 "	52,000 "
4¼ "	95,000 "	50,000 "
4⅛ "	95,000 "	53,000 "
4¾ "	96,000 "	50,000 "
5 "	93,000 "	47,000 "
5¼ "	90,000 "	52,000 "
5⅛ "	90,000 "	53,000 "
5¾ "	90,000 "	50,000 "
6 "		

Experiment to Determine the Effect of Colloidal Copper upon Delaware River Water Germs and upon Bacillus Typhosus.

500 c.c. of Delaware River water collected at Market Street wharf was placed in a copper vessel containing 48 square inches of inside exposed surface, and a similar quantity was placed in a glass vessel, and light excluded from both vessels.

The experiment was conducted at room temperature (75°).

The water when placed in the vessels contained 384,000 germs per cubic centimetre.

DELAWARE RIVER WATER.

Time of count.	Organisms per c.c.
Immediately	384,000
In glass, three hours	240,000
In copper, three hours	18,000

To the water in the copper vessel was then added 3 c.c. of a suspension of typhoid bacilli containing 396,000,000 germs.

Counts were made at intervals of fifteen minutes of the contents of both the copper and the glass vessels.

At the expiration of two hours the typhoid bacilli had entirely disappeared from the water in the copper vessel and the water organisms had been reduced to 5400 per cubic centimetre; while in the glass vessel 150,000 water organisms per cubic centimetre were present.

Time of count.	Copperish water containing 18,000 river-water organisms plus 396,000,000 typhoid germs.	Plain Delaware water containing the 240,000 river organisms per c.c.
At once	288,000	240,000
15 minutes	163,300	250,000
30 "	57,600	220,000
45 "	30,000	200,000
1 hour	13,300	200,000
1¼ hours	10,000	180,000
1½ "	8,000	200,000
1¾ "	7,500 (no typhoid noted).	150,000
2 "	5,400	150,000

Experiment to Determine the Life of Typhoid Bacilli in Water that has been kept in a Copper Vessel in Comparison with that which has been kept in a Glass Vessel.

Water sterilized in an autoclave in a glass vessel was used in this experiment.

A litre of this water was placed in a copper vessel having an inside surface of 79 square inches and another litre of water was placed in a glass vessel and light was carefully excluded.

Both vessels were allowed to stand for three hours at room temperature.

At the end of that period the water in the copper vessel was poured into a glass vessel similar to the control and light excluded.

4 c.c. of a suspension in water of bacillus typhii containing about 10,000,000,000 were placed in each vessel and counts were made at intervals of fifteen minutes for two hours.

The three hours' exposure of the litre of water to the 79 square inches of copper surface was, as previously stated, dissolved, 1 part of colloidal copper to 4,000,000 parts of water.

Time counts were made	No copper present. No. of colonies per c.c.	Copper present 1 part to 4,000,000. No. of colonies per c.c.
At once	7,630,000	3,600,000
15 minutes	7,296,000	2,760,000
30 "	7,800,000	2,400,000
45 "	7,600,000	1,680,000
1 hour	6,100,000	540,000
1¼ hours	4,840,000	480,000
1½ "	4,800,000	180,000
1¾ "	3,040,000	17,000
2 "	2,920,000	0 (16)

The influence of the colloidal copper present is shown most conclusively by this experiment.

Experiment to Determine the Length of time Required for Typhoid Organisms to Die in Copper Vessels, Aluminium Vessels, and Enamel Ware Vessels.

Vessels of the same general shape and size were cleansed with hot water, and 1000 c.c. of sterile water placed in each.

To each vessel was then added 16,875,000 typhoid organisms, and counts were made every half hour.

Time count was made	Copper. Colonies per c.c.	Aluminium. Colonies per c.c.	Enamel. Colonies per c.c.
Immediately	42,800	43,200	42,500
½ hour	6,480	43,000	45,000
1 "	36 (?)	10,800	27,000
1½ hours	0	5,400	27,000
2 "	0	3,800	27,000
2½ "	0	960	21,000
3 "	0	0	20,000

CONCLUSIONS. (1) There is a natural tendency for typhoid bacilli to die when the water containing them is allowed to stand for a long period. There may be a temporary increase in the number, but this is followed in several hours or days by a decrease and a final disappearance.

(2) Trials were made as to the period of total disappearance of typhoid organisms which had been placed in sterile Schuylkill water and in that taken directly from the tap, and from the river surface containing large numbers of water organisms. These waters were placed in vessels of glass, porcelain, tin, and copper and their contents kept at room temperature were plated every fifteen minutes for periods ranging from three to six hours. All the experiments were repeated many times.

(3) Sterile drinking water in clean copper vessels inoculated with typhoid bacilli invariably showed that the bacilli had all perished in one hour. Water similarly treated in tin vessels invariably showed living organisms at the end of twenty-four hours. Water similarly treated in glass vessels exposed to light showed varying results, but in no instance had the typhoid organisms all perished in three hours. Water similarly treated in enamel vessels showed a slight diminution of the number of typhoid organisms in three hours. Water similarly treated in aluminium vessels showed a disappearance of the typhoid organisms in three hours.

(4) Raw tap-water in glass vessels showed an increase in the number of organisms in three hours; occasionally, there was a slight diminution in their number. Raw tap-water in copper vessels in one experiment showed a diminution from 384,000 germs per cubic centimetre to 18,000 per cubic centimetre in three hours. Usually the diminution was not so great. Raw tap-water containing large numbers of river organisms and considerable vegetable

matter, when inoculated with millions of typhoid organisms and placed in a copper vessel, showed that the typhoid were killed off in one and three-fourths hours to two and one-half hours.

(5) Water containing colloidal copper has a more rapid tonic action upon typhoid organisms than upon river-water organisms.

(6) The quantity of colloidal copper given off from a one-litre copper vessel in three hours was one part to four million. This amount killed off the added typhoid organisms in from one and three-fourths to two and one-half hours, and chemical experience has shown that this amount of colloidal copper is harmless when taken into the human system.

(7) In epidemics of typhoid fever water could be purified of typhoid organisms by allowing it to stand in a copper vessel for three hours.

GLIOMA OF THE NOSE.

REPORT OF TWO CONGENITAL CASES.

By J. Payson Clark, M.D.,

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"The glioma is a tumor consisting of proliferated neuroglia (the supporting tissue of the central nervous system) and of bloodvessels which are accompanied by a small amount of connective tissue." Mallory says further, in the article from which the above quotation is taken, that it is found exclusively in the central nervous system of which it forms the commonest tumor, that it is essentially a benign growth, does not give rise to metastases, but is dangerous partly on account of the pressure it exerts, partly from destruction of the nervous tissue it infiltrates. On account of its vascularity, large and small hemorrhages are not infrequent. Mallory also says: "Gliomata of the retina have not been discussed in this article because no one has yet shown by modern differential stains that they occur." In this connection the following quotation from Ohlmacher is pertinent: "Most of the so-called gliomata originating in the retina, especially in children, are in reality a form of round-cell sarcoma with pronounced metastatic tendencies, which glioma lacks." Other writers, as Senn and Bland-Sutton, speak as if the existence of glioma of the retina in childhood were established. Senn mentions glioma as having been found in the acoustic nerve, and, as a heterotopic tumor, in the kidney, the ovary, and the testicle.

There seems to be some difference of opinion, therefore, among writers on this subject, as to whether or not true glioma ever occurs

outside the brain or spinal cord. But no writer denies that the appearance of this growth anywhere except in the regions just mentioned is very rare. In view of these facts, the two following cases would seem to be worth recording as of unusual importance and interest.

CASE I.—Edward K., aged two years, of American parentage, was brought to the throat clinic of the Massachusetts General Hospital in April, 1903, on account of a rounded tumor of the nose, about the size of a robin's egg, which his mother said he had had from birth and which caused considerable deformity, as the photograph shows. The tumor was soft to the touch and resembled very strongly in appearance and consistency a fatty tumor. There was no pulsation in the swelling nor any change in its consistency when the child cried, so that, although its site and general appearance suggested a possible meningocele, it was assumed as reasonably certain that, at the time the child was seen, the tumor had no direct connection with the brain cavity. On looking into the nose, the left nostril was observed to be almost completely obstructed by a pinkish-gray, polypoid growth, the origin of which could not be definitely determined, although its connection with the external tumor could hardly be questioned. A piece of the growth was removed from the left nostril by means of cutting forceps. Hemorrhage was free but ceased quickly. This specimen was submitted to Dr. J. H. Wright, director of the clinico-pathological laboratory of the hospital, who made the following report:

"Microscopic examination of the specimen sent for examination shows that it consists of a piece of tissue partly covered with mucous membrane. The tissue making up the greater part of the specimen consists chiefly of delicate fibrils and peculiar cells and is clearly atypical neuroglia, as shown by its histological appearance and by the staining reaction of the fibrils. This neuroglia tissue near the mucous membrane gives place to submucous connective tissue, but is not sharply marked off from this connective tissue. In fact, it is seen infiltrating the spaces of this tissue after the manner of a sarcoma.

"Diagnosis. As far as the specimen itself goes, the condition is to be regarded as glioma. The glioma may be in continuity with the brain, but it seems to the pathologist more likely that it is associated with a teratomatous tumor in the neighborhood.

"J. H. WRIGHT."

In view of the unusual character of the growth and the uncertainty of the prognosis it was decided to keep the child under observation for a time before undertaking to remove the tumor, if that were eventually thought advisable. The photograph was taken and the mother was told to bring the child again at a stated time. But she never returned, and no answer could be obtained to several letters.

As the case was a much too important one to be entirely lost sight of, the writer finally made a trip to the city where the patient lived, about twenty-five miles from Boston, and after some difficulty

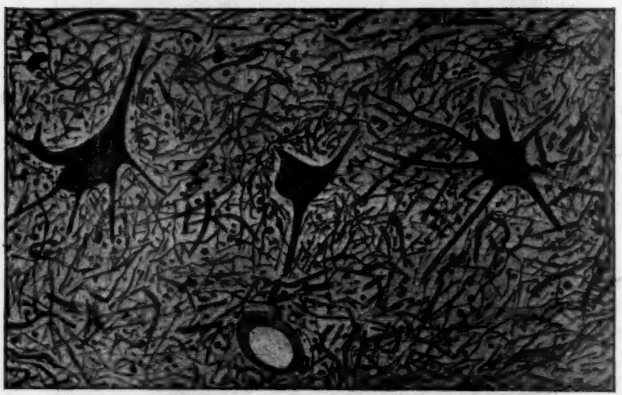
FIG. 1. Photographs of Case I.

owing to the family having moved, found the boy. It was learned that he had been operated on at a local hospital on June 23, 1904. The tumor had evidently been removed, as the nose was normal

except for a rather unusual breadth and a rather prominent linear nearly vertical scar, about 2 cm. in length. The general health of the child appeared excellent, and there was no local evidence of any new-growth. Communication with the hospital where the tumor had been removed elicited the facts that it had not been preserved and that no microscopic examination had been made of it. Nor could any description of the gross appearances at the operation be obtained. Interesting as these reports would have been in completing the history of this case, the diagnosis is sufficiently established by Dr. Wright's report.

The second case was first seen at the throat clinic of the Massachusetts General Hospital on December 21, 1903, by Dr. Frederic C. Cobb, who was then on duty, and he very kindly referred it to me.

FIG. 2.



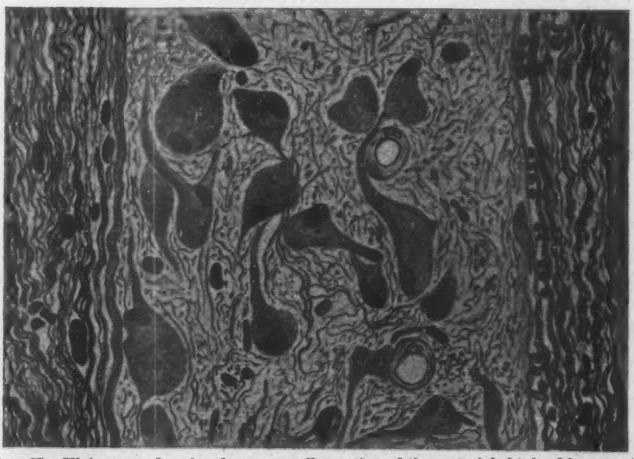
CASE I.—High-power drawing from a paraffin section of the material removed by operation, showing the character of the tumor tissue, which is composed of fibrils and "spider" glia cells.

CASE II.—William A. M., aged ten weeks, was brought to the hospital on December 21, 1903. The father said that the child had not breathed well through the nose since birth and that he had noticed something in the left nostril. The infant appeared in excellent health and took its nourishment well except for the inconvenience caused by partial nasal obstruction. On examination a pinkish-gray polypoid mass was seen in the left vestibule, causing almost complete obstruction of that side. The site of the growth was higher up in the nose, and, as nearly as could be discovered, owing to the difficulty of seeing into so small a nostril, appeared to be the septum. A piece was snared off for microscopic examination. Hemorrhage was free but easily stopped by slight pressure. The pathologist's report on this specimen and on another removed in February, 1904, was as follows:

"On December 21, 1903, and on February 29, 1904, small pieces of tissue were excised from the lesion on the nasal septum. Microscopic examination of both these specimens shows, in the situation of the submucosa of the part, masses of a peculiar tissue which has the structure and staining reaction of a gliomatous tissue. The tissue consists of cells and fibrils in varying proportions. Some of the cells are quite large, have eccentric nuclei and fibrillary processes. This gliomatous tissue in places appears to infiltrate and distend lymph spaces, and columns of it may be seen in the submucosa very near the mucous membrane proper.

"J. H. WRIGHT."

FIG. 3.



CASE II.—High-power drawing from a paraffin section of the material obtained by operation, showing part of a column of gliomatous tissue infiltrating the submucous connective tissue. The tissue, composed of the paler fibrils and the large pyriform cells occupying the middle portion of the drawing, is the gliomatous tissue. The darker fibrils on each side of the drawing represent the connective tissue of the submucosa.

Another specimen was removed on April 20th, and was reported to be practically the same as the first. No photograph of this patient was taken because there was no external deformity. At the date of writing this paper, December, 1904, this child is in excellent health and there is no apparent increase in the growth.

These are certainly genuine cases of glioma outside the central nervous system. They resemble each other closely in every particular except that in one there was an external deformity. They were both evidently of prenatal origin. It is unfortunate that no record of the operation in Case I. could be obtained, for it might have cast some light on the source of the tumor. The writer is inclined to the opinion that these growths arose, during the process of fetal development, through the shutting out of some neuroglia

or, more exactly, that embryonic tissue which becomes neuroglia from the brain cavity in the coming together of the two lateral halves of the frontal bone. In both cases, as far as the histories go, the tumor appears to be absolutely benign. In the first case the tumor had not changed in size from birth up to two years of age, according to the mother's observation. It was removed six months ago, nearly, and there is no sign of recurrence. It is about a year since the other case was first seen. During this time the growth has shown no apparent increase. The writer will endeavor to keep in touch with these cases, and if there are any new developments he will report them. A thorough search of medical literature for a period of more than ten years past reveals no other reported case of glioma of the nose.

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A CASE OF CEREBROSPINAL RHINORRHŒA, WITH RETINAL CHANGES.

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AND
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A REPORT of this case is important as a contribution to the collected cases of a very rare affection and likewise as an additional evidence of the importance of the eye-ground changes in obscure conditions affecting the central nervous system. In 1899 Dr. St. Clair Thomson published an elaborate and altogether admirable monograph which contains the fruits of a painstaking search through medical literature for instances of a curious affection of which the most conspicuous symptom is the spontaneous escape from the nose of cerebrospinal fluid. He succeeded in unearthing the clinical records of 20 cases in which a watery flow from the nostril could with reasonable certainty be regarded as originating from the cranial cavity. Many cases in which the predominance of evidence spoke for other possible sources of the watery flow—*e. g.*, nasal hydrorrhœa, dropsy of the antrum, serous accumulation in the maxillary sinus, vasomotor neuritis, and ruptured lymph tubes —were carefully differentiated and excluded.

These 20 cases, then, or 21 including the case investigated by Thomson, may properly be regarded as instances of this curious affection. In commenting upon his monograph the *British Medical Journal* has this to say, and in no better way than by quoting from its editorial can the debt which medicine owes to Dr. Thomson for his careful study of this little-understood disease be expressed: "From the dust-heaps of medical literature he has been able to unearth the details of no fewer than 8 undoubted cases similar to his own and 12 others which were probably of the same character. These cases were, as a rule, described and indexed under different titles. In addition, in the works of Willis and Morgagni, there are records of others which are suggestive of this condition; so that cerebrospinal rhinorrhœa is no new disease, but one which, though undoubtedly rare, may not be so uncommon as would at first be thought."

That the disease is indeed rare can be seen from the few reported cases and also from the fact that, in a personal letter to the authors, St. Clair Thomson states that, though constantly on the lookout for similar cases, his published case remains the only one in his experience. His large clinic at Kings College Hospital and the Throat Hospital has in over five years yielded no additional example of this affection.

At the time of Dr. Thomson's report such a case had an interest that at present does not exist. The escape of cerebrospinal fluid from the nose in quantities sufficient for purposes of examination gave an opportunity for the study of this fluid which up to that time did not often present itself. At the present time it is always an easy matter to obtain a sufficient amount by lumbar puncture for all purposes of laboratory study. It is scarcely to be wondered at that such men as Halliburton, Hill, and others should have made elaborate studies of the fluid from Thomson's case in regard to the chemical, bacteriological, and morphological contents of the collected specimens. Their work has made it possible to determine in a given case whether the fluid so obtained is cerebrospinal or simply a secretion from the nose due to other causes.

A certain number of the cases which have been reported and those contained in the original monograph have been accompanied by optic neuritis and optic atrophy. Of the 20 cases discussed by Dr. Thomson, 8 presented more or less severe affections of the nerve-head and retina adjacent. These 8 cases, which are probably all the cases in literature of cerebrospinal rhinorrhœa with ocular complications up to 1899, together with Freudenthal's and the present case, have been tabulated by us with reference to vision, the state of the pupils, the condition of the fundus oculi, the visual and color fields, the time relation of the ocular symptoms and the nasal flow, the nostril from which the fluid escaped, and miscellaneous ocular symptoms:

Author.	Vision.	Pupils.	Fundus oculi.	Visual fields.	Time relation of ocular symptoms and nasal flow.	Flow from right or left nostril.	Other ocular symptoms.
Leber. Von Graefe's Arch f. Ophthalm., 1883, vol. xxix., Heft 1, p. 273.	O. D. V., fingers. O. S. V., hand motion. 4 yrs. later, O. D. V., 0. O. S. V., fingers.	React promptly.	Right disk blanched, papillary and retinal vessels narrowed. L. disk blanched, nasal border indistinct, smaller veins tortuous	Greatly contracted except in upper vertical meridian.	Flow 5 years after failure of vision.	Left.	Color blindness.
Mackenzie. Trans. Intercolonial Med. Congress of Australasia, Third Sess. Sydney, 1892, p. 500.	V., p. l., both.	Partly dilated, equal, react slightly to light.	Optic neuritis, both; three years later, optic atrophy.	....	Flow 1 year after failure of vision.	Left.	
Körner. Zeitschr. f. Ohrenheilk., July, 1897, Bd. xxxiii., Heft 1.	O. D. V., $^{5}/_{9}$. O. S. V., fingers.	R. normal, good light reaction. L. dilated, no reaction to light; reaction to accommodation present.	Optic atrophy, both.	.....	Flow several years after failure of vision	Left.	Slight prominence of globe; rotatory nystagmus; insufficiency of interni.
Baxter. Brain, January, 1882, vol. iv. p. 525.	O. D. V., $^{12}/_{60}$. O. S. V., $^{12}/_{60}$. Later vision failed nearly to blindness.	Equal, react well.	Optic neuritis, both.	Incomplete hemiopia, upper and outer quadrant of each visual field defective.	Flow 2 years before failure of vision.	Right.	Color perception normal.
Nettleship. Ophthalmic Review, 1883, vol. ii. p. 1.	O. D. V., $^{20}/_{100}$. O. D. V., $^{20}/_{70}$ with $\bar{}$ —1 sph. $\frown$ —1.25 cyl. O. S. V., $^{20}/_{0}$, not improved.	Large and sluggish.	Postpapillitic atrophy, veins tortuous, vessels shrunken.	Greatly contracted, green field very small; in better eye field for red about equal to white field.	Flow 1 year after failure of vision.	Left.	Color perception normal.
Priestley Smith. Ophthalmic Review, 1883, vol. ii. p. 4.	O. D. V., faint p. l. O. S. V., 0.	.....	Disks atrophied.	.....	Flow 4 years after failure of vision.	Left, later right.	Horizontal nystagmus; R. eye making larger excursion than L. Nystagmus.
Priestley Smith. Ibid., p. 7.	V. both, 0.	Dilated.	Double optic neuritis, later atrophy.	.....	Flow 2 years after failure of vision.	Right, later left; right nostril occluded by polyp.	
Emrys-Jones. Ibid., 1888, vol. vii. p. 97.	O. D. V., $^{6}/_{6}$. O. S. V., fingers to outer side.	.....	R. Disk pale, atrophic L. well-marked atrophy.	Marked concentric contraction.	Flow 10 years before failure of vision.	More discharge from left than right.	Color perception normal.
Freudenthal. Virchow's Archiv, Bd. clxi. Heft 2, p. 328.	O. D. V., $^{20}/_{30}$. O. S. V., $^{20}/_{100}$.	React well.	R. mild grade of papillitis. L. neuroretinitis.	.....	Flow 6 months before failure of vision.	Left.	
Authors' Case.	O. D. H. 1.25, V. $^{16}/_{16}$. O. S. H. 1.25, Ah. 0.75, Mc. 90, V. $^{16}/_{24}$.	Equal 4 mm. diameter, react sharply to light and accommodation.	Partial postpapillitic atrophy, both.	Moderate concentric contraction for white, more marked concentric contraction for blue, red, and green.	Flow 4 years after slight failure of vision.	Right.	Color perception normal.

In all cases the ophthalmoscopic pictures were strikingly similar: either a full-blown optic neuritis passing into atrophy, or typical postpapillitic atrophy. The pupils were usually dilated, reacting feebly to light stimulation. In 5 cases the visual fields were examined and found contracted. The only instance of examination of the color fields was in a case reported by Nettleship, who found the field for green very much contracted, while that for red almost equalled the white field. Central color perception was found normal in 4 cases; color blindness in 1. The severity of the inflammatory process and its destructive effect on the fibres of the optic nerve may be judged from the fact that the visual outcome in 5 cases was bare perception of light or total blindness. In the remainder, one eye, although presenting indubitable ophthalmoscopic evidence of participation in the inflammatory process, still retained a fair measure of vision. In the case reported by Emrys-Jones the better eye attained a vision of 6/6 after correction of its ametropia. Nystagmus was observed in 3 cases. Slight prominence of the globe and insufficiency of the interni were noted in 1 case (Körner).

In 4 cases it will be observed that the greater visual defect obtained in the eye on the same side as the nasal flow. Failure of vision preceded the establishment of the flow in 7 cases, the interval being from one to five years. In 3 cases the flow began several years before the onset of ocular symptoms.

CASE.—Miss Z., servant, aged thirty-two years, was referred to Dr. Schwab by Dr. H. W. Loeb, the laryngologist, for examination of her nervous system. Her chief symptom was a continuous watery discharge from the right nostril in the presence of an absolutely negative nasal and throat condition. Her family history indicates a definite neuropathic stock. One sister died in an insane asylum. Two brothers are living, of whom one shows many of the symptoms of the patient other than the nasal ones. One sister died of tuberculosis. The patient has always been considered of a nervous temperament, and she herself has been aware of this condition. Her history shows several attacks of illness of a vague nature, from which she recovered slowly under a more or less systematic rest. None of these attacks show any trace of an infectious or organic origin. Seven or eight years ago there was a period of a year or more during which she was not able to work for any length of time, yet with no definite symptoms other than fatigue. She was easily tired, had headaches frequently, and was unable to sleep. Rest and a vacation afforded relief. About four years ago she was suddenly attacked by a paralysis of the left leg, which compelled her to remain in bed for some weeks. The leg was not painful, but was stiff and weak. From this she recovered fully, no trace remaining. The present trouble began about two years ago, following a cold, or at any rate the patient first noticed at this time the discharge from the nostril. The fluid dropped continuously

from the nostril day and night. The amount varied from time to time from a few drops to 3 or 4 c.c. in an hour. When the patient is worried or fatigued the flow increases. The fluid is clear, tasteless, and does not stiffen or stain the handkerchief. If the patient reads or inclines the head to the right the amount of the escaping fluid is increased. There is no pain or any discomfort other than the necessity of always taking care of the discharge. Occasional headaches, of which the patient complains, are not in any way influenced by the amount of discharge. Six years ago the patient had an attack of "pink eye," recovering promptly under treatment. In 1899 she noticed some haziness of vision, accompanied by symptoms of asthenopia. The report obtained from the oculist who treated her at that time is as follows: "Neuroretinitis; swelling of 3 D. in the right disk; left disk, beginning atrophy."

The physical examination of the internal organs shows no abnormality. The nervous system gives evidence of a neurasthenic-hysteric group of symptoms, not particularly well marked. The knee-jerks are increased, as are the plantar and Achilles reflexes. The Babinski reflex is not present. The sensory system shows some anomalies. The conjunctiva and the pharyngeal mucosa are anæsthetic. There is a marked hyperæsthetic area in the small of the back, which is very tender to the slightest pressure. The fingers and hand show a very marked tremor, coarse and slow, not affected by rest or motion. The urine is normal with the exception of a rather high specific gravity. As all the specimens examined showed this high specific gravity, it is probable that the continual leakage of fluid from the nostril accounts for it. The patient is distinctly hypochondriacal, much interested in her condition, for which she is convinced a disease of the spinal cord is responsible.

Many specimens of the fluid were examined, collected under different conditions, and in varying amounts from a few drops to several cubic centimetres. There was little difference in the specimens so studied. The fluid is clear, odorless, and tasteless. Specific gravity about 1010. No albumin and no mucin were found. In one specimen there was obtained a reduction by Fehling's test. Centrifugalized specimens showed, beyond a few leukocytes, nothing abnormal and no other formed elements.

Ocular Examination. Pupils equal 4 mm. in diameter. Direct light reaction sharp. Consensual a little sluggish. Reaction to accommodation present. Ocular movements unrestricted in all directions. No tendency to outward deviation of either eye during strong convergence. With Maddox rod, esophoria 2°, no hyperphoria. Nystagmus absent. The accommodative power is normal for the age of the patient. The refraction, estimated under homatropine cycloplegia, proved to be: O. D.—H. 1.25 D.; V = 16/15. O. S.—H. 1.25; Ah. 75; Mc. 90; V = 16/24. Color perception normal. No central scotomata.

Ophthalmoscope. Right eye: Disk generally pale; outer margin clearly outlined, inner a little blurred. Veins moderately full and slightly tortuous. Arteries distinctly narrowed and "corkscrewy." Grayish stippling of retina around disk. Macula and periphery of fundus normal. Left eye: Disk blanched, the outer quadrant occupied by a glistening white, semicircular area. Veins narrowed; arteries much contracted and "corkscrewy." Extensive grayish stippling of retina surrounding disk. Macula and periphery of fundus normal.

The charts show a moderate concentric contraction of the visual field for white and a more marked concentric contraction of the color fields. It will be noted that the degree of peripheral defect is greater in the left eye, which, visually and ophthalmoscopically, has evidently sustained the brunt of the inflammatory mischief.

The dropping from the nose first observed in April, 1903, has always been from the right nostril. It did not appear, therefore, until four years after the onset of the neuroretinitis. The greater degree of atrophy is found in the nerve on the side opposite the dripping nostril. In both respects the case offers a contrast to the majority of the cases in the table.

The feature in which the present case differs conspicuously from the clinical picture of hitherto recorded cases of cerebrospinal rhinorrhœa consists in the preservation of a high and almost equal degree of visual acuity in both eyes, despite indubitable evidence of inflammatory and atrophic changes in both optic disks. It should be borne in mind, therefore, that while the type of optic neuritis in this disease is usually severe and greatly destructive of sight, a much milder form (Baxter, Nettleship, and the present case), running a typical inflammatory course without seriously impairing the fibres of the nerve, is occasionally encountered. Wholly unimpaired central visual acuity cannot justify the assumption that there is no disease of the optic nerve. A rigorous ocular examination, with especial reference to the visual and color fields, may disclose the presence of optic-nerve mischief in a majority if not in all cases of cerebrospinal rhinorrhœa. This phenomenon should then properly be regarded as an integral part of the symptom complex and not merely as a complication.

Since the publication of Dr. St. Clair Thomson's monograph, only one case of this condition associated with optic neuritis has, so far as we can ascertain, been recorded. Freudenthal describes a case of watery flow from the left nostril in which an optic neuritis developed eight months later. Vision reduced to O. D. 20/30; O. S. 20/100. The ophthalmoscope showed a mild grade of papillitis in the right eye and a frank neuroretinitis in the left.

The diagnosis of the case as one which properly belongs to that class of cases which St. Clair Thomson reports cannot be in much doubt. The chief question at issue is the decision in regard to the

CHART I.

Left. White. Right.

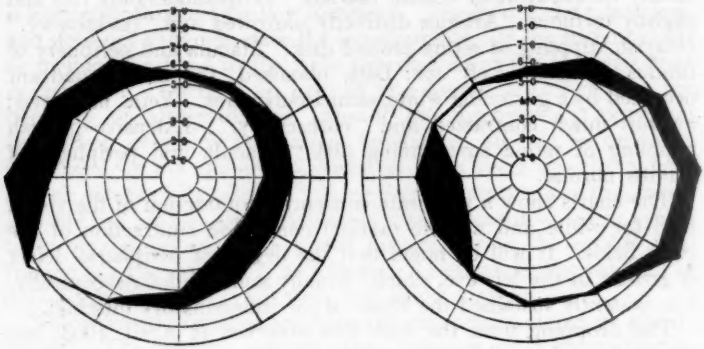


CHART II.

Left. Blue. Right.

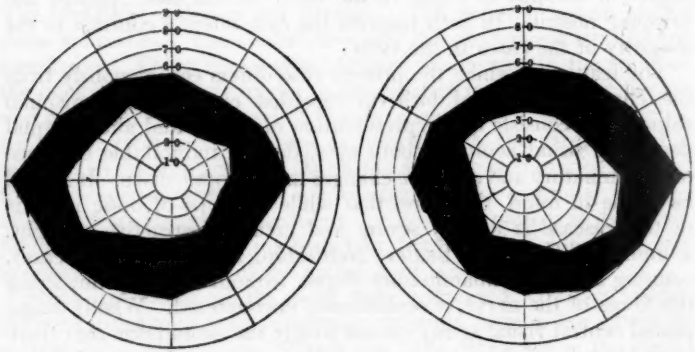


CHART III.

Left. Red. Right.

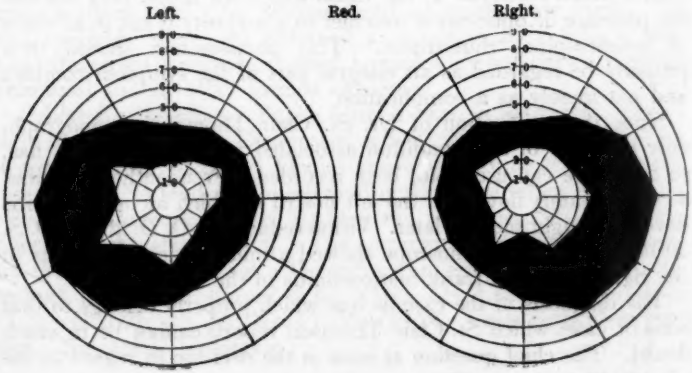
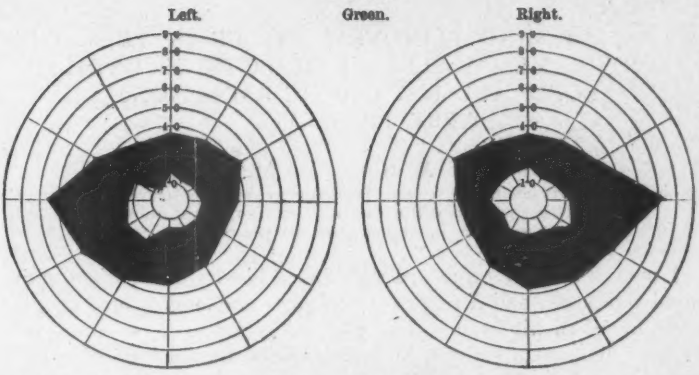


CHART IV.



fluid. If it can be said to be cerebrospinal in character, then the diagnostic problem is solved. That it is possible to determine this with absolute certainty is due to the work of Halliburton and Hill, as was previously pointed out. Halliburton distinctly says that this fluid stands apart from all other similar fluids. "The diagnosis of cerebrospinal fluid from other forms of watery flux from the nose need not in future present any difficulty. The constant and long-continued escape from the nostril of a perfectly clear watery fluid should always arouse a suspicion as to its subarachnoid origin. When this fluid is found to be free from taste, smell, and sediment; when albumin and mucin are found to be practically absent from it, and when Fehling's solution is reduced by it, the suspicion becomes a certainty."[1]

In regard to the etiology of this strange affection we can add nothing of importance. Whether it is due to some inflammatory process or whether it is in part due to certain anatomical defects in the skull are at present open questions. The presence of the ocular changes are, therefore, of great importance because they seem to point to the fact that there must have been some general process of perhaps an inflammatory or circulatory character which first made itself evident in the optic nerve and then afterward gave rise to the other symptom, namely, the release of cerebrospinal fluid from the skull through the nose. The assumption that there is in some of these cases, particularly the one reported by Freudenthal, a cerebral tumor which is the causative agent we believe to be unfounded.

The point which we especially desire to emphasize is that the ocular changes in these cases are probably not accidental, but are an integral part of the disease of which they form one part and the escape of the cerebrospinal fluid forms the other.

[1] British Medical Journal editorial.

A CASE OF MIND BLINDNESS UNIQUE IN THAT THE ENTIRE MESIAL SURFACE OF BOTH OCCIPITAL LOBES AND BOTH OPTIC RADIATIONS WERE PRESERVED.[1]

By Ward A. Holden, A.M., M.D.,

Instructor in Ophthalmology, Columbia University; Consulting Ophthalmologist to Bellevue Hospital, and Ophthalmologist to King's Park and Ward's Island, W. State Hospitals for the Insane.

I desire to place on record the clinical history of a patient with mind blindness, and a report of the gross and microscopic conditions found in his brain. The case is, so far as I can discover, unique; the patient suffered for a long period from a form of blindness, yet no lesion was found on the mesial surfaces of the occipital lobes or in the optic radiations, as is the rule in cases of mind blindness. But symmetrical areas of softening were present in the outer cortex of each cerebral hemisphere, including on each side the angular and supramarginal gyri. I am indebted to Dr. Wilgus for the patient's history while in the King's County Hospital, to Dr. Simon for his history while in the King Park Hospital, to the late Dr. Williams for the autopsy report, and to Dr. C. B. Dunlap, of the New York State Hospitals' laboratory, for excellently prepared sections of the brain, eyes, and optic nerves.

W. S., male, aged fifty-three years, was seen by me at the Long Island State Hospital for the Insane at King's Park, October 19, 1902. His pupils were of medium size and responded promptly to light, and his fundi were normal. He was, however, apparently unable to count fingers and his eyes did not follow a light. Nevertheless he seemed to me to be visually aware of the presence of persons in the room, and although at first glance he impressed one as blind, the ward attendants were sure that at times he saw, and his behavior differed from that of patients in the same ward blind of optic nerve atrophy.

My frequent examinations revealed no changes in the pupils or fundi up to the time of his death, August 26, 1903, ten months later.

At this point I might say that the retinæ and optic nerves when examined microscopically were found to be normal.

When I first saw the patient he was demented, aphasic, and extremely obstinate, so that extended psychical examinations could not be carried out, and hence I was largely dependent upon the records of hospitals in which he had been previously and upon the anamnesis furnished by his wife, an intelligent woman.

[1] A preliminary report of this case was read before the American Ophthalmological Society, July, 1904.

The chief points in his history are as follows: Eight years before his death he staggered one day while in the street, and after reaching home was found to be numb on one side and unable to speak intelligibly. The following day he seemed quite well again and returned to his work—that of roasting coffee.

In March, 1902, seventeen months before his death, he came home one night feeling ill. On arising the next day he bumped into objects, apparently being blind. His mind was confused and he was unable to speak intelligibly. He went to bed and remained there a week, after which he was able to get up, but his left arm and leg were very weak. He had become emotional and depressed. He refused to take objects handed to him, or to feed himself, or to help himself in any way. He accused his wife of infidelity and was ugly, refusing to answer her questions.

In June, 1902, with his wife he visited his mother-in-law. At her house improvement was noticed and at times he talked quite rationally with his mother-in-law, whom he liked. Speech was difficult and memory for recent events was very poor. He seemed to see, but he never tried to read. When he walked his wife always led him because of his muscular weakness, and therefore it was difficult for her to judge of his powers of orientation.

At this time he had frequent visual hallucinations, believing, for example, that he saw men in the street when no one was there.

In July, 1902, he failed both physically and mentally, and then ceased to answer questions. On some days he bumped into objects, on others he seemed to see. He talked thickly and besides this failed to recall the words he wished to use. He was fond of smoking, but now when he took his pipe in his hand he was unable to fill it or even to hold it properly, not knowing whether he was grasping the bowl or the stem (apraxia), and he puffed away contentedly when an empty pipe was placed in his mouth, provided that a match had been struck and held near it.

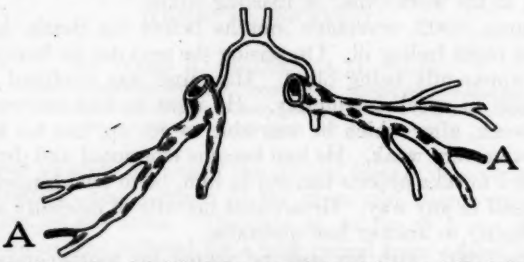
In the King's Park Hospital he slowly wasted away, and died finally of enteritis. His wife frequently visited him, and up to the last she thought that on some days he saw, since he would answer correctly when she asked him if she had on her hat or gloves, or some question of this sort. On other days he refused to answer such questions, and on these days she believed him to be blind.

To sum up, this was a case of dementia after hemiplegia, together with aphasia, apraxia, and an interference with vision which much of the time amounted evidently to total blindness.

There are doubtless several forms of mind blindness. This case was apparently not of the commonest form, which has been much studied in recent years, particularly by Liepmann. In the common form of mind blindness the patient unquestionably sees but does not recognize what he sees. That is, perception takes place probably in the lower cortical visual centre about the calcarine fissure,

but there is destruction or cutting off of the higher centres of visual apperception which lie presumably in and about the angular gyri. With this form of mind blindness there are, as a rule, hemianopic

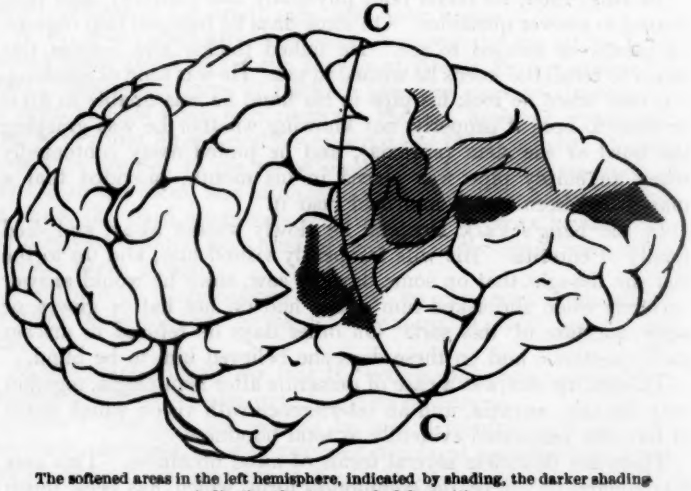
FIG. 1.



The internal carotids and their branches, sclerotic in patches. *A A*, blocked parieto-temporal branches of the middle cerebral arteries.

defects in the visual fields, and frequently aphasia and also astereognosis—the inability to recognize objects by the touch, or apraxia—the inability to handle objects recognized. The patients have arteriosclerosis, and occlusion of cerebral arteries leading to softening in the occipital lobes is the anatomical cause of the

FIG. 2.



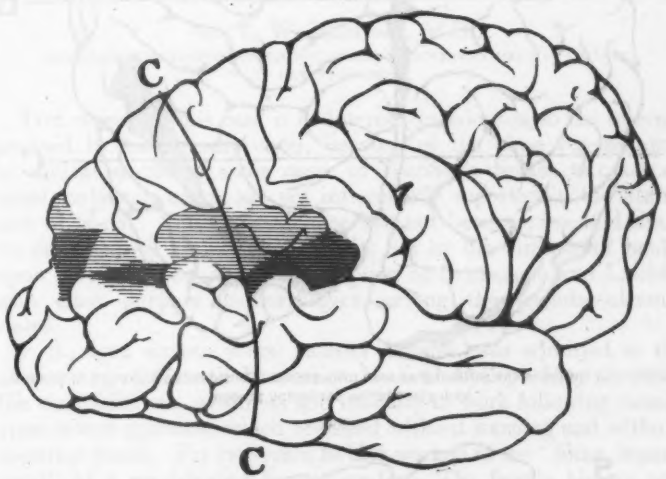
The softened areas in the left hemisphere, indicated by shading, the darker shading indicating the more marked destruction.

mind blindness. In the cases reported, so far as I can learn, there has always been found, besides other lesions, either destruction of the cortex near the calcarine fissure on the mesial surface

of the occipital lobe, or destruction of the optic radiation. Yet there are cases on record in which there was bilateral destruction of the calcarine cortex without true mind blindness; hence it has been supposed that the cortical centres whose destruction or cutting off cause mind blindness lie outside of the cuneus.

In this case the vessels of the brain exhibited many patches of sclerosis and the parieto-temporal branch of each middle cerebral artery, which runs in the fissure of Sylvius, was occluded from the point where it was given off (Fig. 1, *A A*). This occlusion had caused softening in an extensive area in each hemisphere, including the angular and supramarginal gyri, and reaching back on each side nearly to the tip of the occipital lobe (Figs. 2, 3 and 4). There was,

FIG. 3.



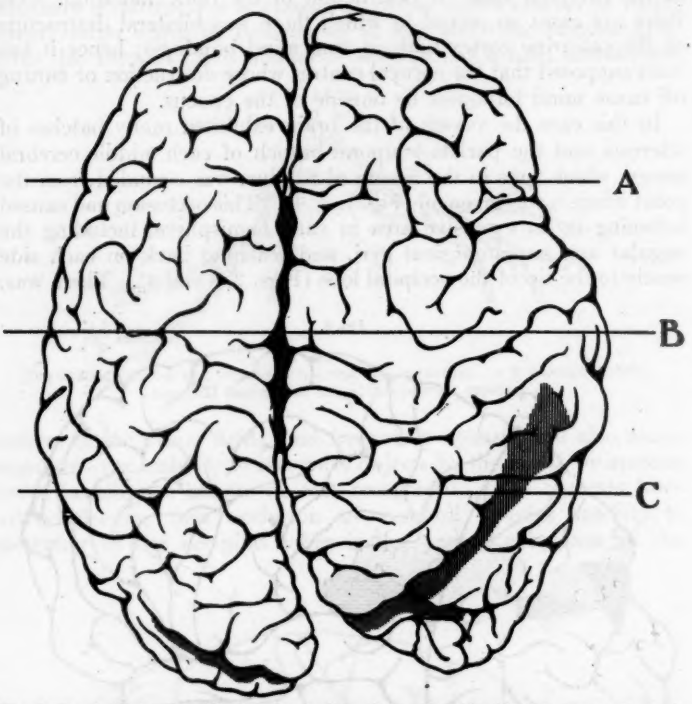
The softened areas in the right hemisphere.

however, no softening on the mesial surface of the occipital lobes, and in a frontal section 4.5 cm. anterior to the tips of the occipital lobes the softening of the outer cortex on each side was found to extend only to the upper margin of the tract of white matter whose lower portion is supposed to contain the optic fibres (Fig. 5, *W W*).

In microscopic sections the softening extended no deeper than is shown in Fig. 5. And in serial sections 1 mm. apart stained by Weigert's method the visual fibres which run from the external geniculate bodies through the optic radiations were not degenerated. In other words, the connection between the external geniculate body and the calcarine cortex on each side was unbroken.

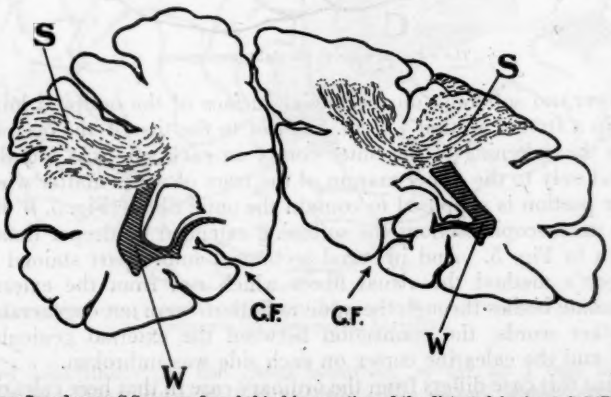
Thus this case differs from the ordinary case in that here calcarine cortex and optic radiations were preserved. Here the primary visual

FIG. 4.



The shading indicates the softening as seen from above. Considerable distortion of the brain took place in the hardening process.

FIG. 5.



The softened areas *S S* as seen from behind in a section of the distorted brain at *C C* (Figs. 2, 3 and 4): *C F*, calcarine fissures; *W W*, the tracts of white matter which include the optic radiations.

pathways from retinæ to calcarine fissures were normal. But softening of the angular gyri and the parts adjacent, in which it is believed the higher visual centres lie, produced such marked disturbance of vision that it was a question much of the time whether the patient had any power of visual perception.

This case then shows that extreme disturbance of vision may be brought about by lesions in the higher cortical visual centres alone, to which we ophthalmologists have, as yet, given little attention.

PURPURA HEMORRHAGICA.

By T. W. Hastings, M.D.,
INSTRUCTOR IN CLINICAL PATHOLOGY, CORNELL UNIVERSITY MEDICAL COLLEGE, NEW YORK CITY.

The report of this case is of interest, in addition to the interest aroused by rather rare cases, because of the close resemblance in clinical course of some cases of Werlhof's disease to cases of acute leukæmia which are not infrequently reported in the literature of to-day. This resemblance has not been commented upon by most writers on acute leukæmia, nor by the authors of books upon blood diseases, with the exception of Benzançon and Labbé,[1] who class purpura hemorrhagica among the pseudoleukæmic states.

J. S., aged sixteen years, nativity Ireland, was admitted to the Dispensary of Cornell University Medical College May 3, 1902. His complaint was weakness and inability to work following closely upon severe epistaxis, which occurred without warning and without apparent cause. For two years he had worked in the "filing department" of a neighboring brass foundry. The family history was satisfactory, including no "bleeders," and neither deaths nor sickness suggesting the less common diseases; he was one of six healthy children.

The history of the patient's early years, obtained from his mother, was that of a healthy, active boy who escaped the exanthematous and infectious diseases of infancy and childhood, apparently without a day's illness until two weeks before coming under observation. For one year the boy had smoked cigarettes and a pipe, but not to excess. There was no history and no evidence of venereal disease. Rarely a glass of beer was taken. The usual diet consisted of ham, eggs, potatoes, tea, toasted bread, with fresh meat and other vegetables at least twice a week, and under treatment

[1] Traité d'hematologie, 1904, p. 683.

the substitution of fresh meat and vegetables and fruit did not influence the course of the disease.

Twelve days before admission he stopped work on account of increasing weakness, which was first noticed the day following a severe "nose-bleed," fourteen days before admission. During the six days following the initial severe epistaxis slight bleeding continued "by spells" each day, and at this time the pallor was noted and the spitting of blood occurred for the first time. No soreness of the mouth was complained of and the parent and the boy thought the latter bleeding from the lung, although on admission the condition of the gums would have accounted for a moderate amount of "blood spitting." The urine was not noticed during this period of two weeks, and had been previously always "clear and yellow;" but twelve days before admission the stools were noted as black and sticky, and the same description was applied later to the stools, which were found to contain much blood and blood pigment.

At no time were there chills, rigor, headache, nausea, or vomiting to suggest the onset of an acute infection. There were loss of appetite and loss in weight.

Physical Examination. Fairly well nourished boy. Facies sickly, pale, a yellowish pallor, ears bloodless; dark-red, crusty particles about nares and corners of mouth. Conjunctivæ pale. The tongue was pale and covered with a black coating, which he stated was from his medicine (iron). The right tonsil was swollen; the left tonsil normal; no swelling of left or right cervical glands. The gums were soft, but only slightly swollen, and from them constantly oozed thin, watery blood. A few small blebs, ½ mm. in size, were on the right eyelids. Temperature by mouth 101°.

Thorax. Thin and shallow, symmetrical; expansion fair.

Lungs percussed normally, and sounds were normal over right and left fronts and backs.

Heart. Maximum impulse in fifth space well inside nipple line; area normal. Over the point of maximum impulse and extending inward and upward toward the pulmonic region was heard a soft, systolic, blowing murmur, greatest intensity at the apex; a similar murmur in the second and third left spaces over the pulmonic area. The pulse was 120 to the minute, small and regular.

Abdomen. Soft, not tender; stomach area normal; spleen not palpable and area not enlarged by percussion; liver area normal, edge not palpable in right midclavicular line. No rose spots.

Hemorrhagic Eruption. Scattered over the upper thorax, back and front, and the neck were a few small, irregular spots suggesting bruised areas, varying in color from a purple to a yellow color. No petechiæ, and no fresh cutaneous hemorrhages. Over the sacrum was a large (10 x 13 cm. = 4 x 5 inches) purple-red purpuric spot which had appeared twenty-four hours previously, tender on pressure, but no tumor and no induration. Over the external

surfaces of both thighs just above the knees, over the legs just below the knees, and over the lower portion of the legs were scattered numerous, irregular, purpuric spots of a greenish-yellow color, varying in size from 1 to 4 cm. in largest diameter. No history of injury. No œdema about ankles.

Rectal examination was negative. No hemorrhoids and no evidence of infection about anus or in lower rectum.

Genitalia. Puerile; otherwise normal.

Glands. No glandular enlargement.

Blood Examination, May 3, 1902. Coagulation time seven to nine minutes (Wright). Hæmoglobin, 33 per cent. (von Fleischl); red blood corpuscles, 1,916,000; white blood corpuscles, 3000.

Malaria. Parasites not found.

Red cells fresh and stained appeared normal, excepting moderate polychromatophilia; no erythroblasts. Differential (400 cells): polynuclears, 50.3 per cent.; lymphocytes, 38.3 per cent.; large mononuclears, 11.3 per cent.

Urine. Clear; light yellow; 1012; albumin, 0; glucose, 0; red cells, 0.

May 6, 1902. To-day the boy is pale, anæmic, facies typhoidal. Tongue slightly furred, edges coated, tremulous. Temperature, 102.8°; pulse small, regular, rapid, 150 to minute. Right tonsil no longer swollen, and not red. Cervical glands not swollen. Gums not spongy. The patient's mother stated there had been "no bleeding during the three days since first visit, but yesterday the bowels moved every half-hour and the movements were black and watery," and the patient complained of pains in the abdomen.

8*th.* The patient's mother visited the dispensary and reported that "his throat was sore," and that there was some swelling on left side of neck; the pulse quiet and slow and regular; the stools natural; the urine clear and yellow. The "black and blue" spots were still present.

The parents would not permit entrance to a hospital, even though unable to keep the boy in bed. The swelling on "left side of neck" proved to be on both sides, due to the heaving-full pulsation of cervical vessels so often seen in severe anæmia.

13*th.* The purpuric spots had disappeared. Color pale lemon yellow, similar to that of pernicious anæmia in adults. Gums pale, firm, and no longer bleeding. Temperature, 100°; pulse, 96, soft, fair, volume regular. Has spat up a small amount of bloody sputum, but no dark stools and no dark urine during the last few days. Throat no longer sore. No enlarged glands in neck.

17*th.* He visited the dispensary. The pallor was marked, the eyelids purple, conjunctivæ pearly. Tongue and throat clear and normal. Temperature, 100°; pulse soft, regular, not dicrotic, 140 to minute. *Heart* showed no enlargement. Apex in fifth

space well inside nipple line, 8 cm. from midsternum. Systolic murmur over apex and base.

No definite spots over trunk or limbs, but a peculiar bluish, mottled appearance over upper thorax and shoulders in front. No glandular enlargement. No enlargement of spleen.

Blood Examination. Coagulation time three and a half minutes (Wright). Fresh blood: red cells pale. Plates practically absent. Hæmoglobin, 20 per cent. (von Fleischl); red blood corpuscles, 1,300,000; index, 0.76; white blood corpuscles, 4000.

Red cells: slight poikilocytosis; anisocytosis absent; polychromatophilia slight; erythroblasts not found. Differential (500): polynuclears, 22 per cent.; lymphocytes, 66.5 per cent.; large mononuclears, 11.5 per cent.

Serum Reaction. Widal's negative 1 to 60 in three hours.

19*th. Urine Examination.* Pale yellow; clear; acid; specific gravity, 1010; albumin, 0; sugar, 0; casts, 0; red cells, 0. Spectroscopic examination: No blood derivatives.

On May 27, 1902, death occurred. Two days preceding death large purpuric spots appeared over the dorsum of the left foot and about the ankles; the feet and ankles and legs became œdematous, and there was a thin, bloody discharge from the mouth and nose.

The case was treated unsatisfactorily, in that hospital care was not permitted and the patient could not be confined to bed at home. Tincture of iron chloride, iron tonics, arsenic, together with regulation of diet, constituted the treatment. Adrenalin was given for two days from May 6th, and apparently affected the melæna favorably, while later the same drug failed to affect the bleeding from gums and bowel. The slight sore throat was not complained of until after the first inspection, May 3d, although at that time the right tonsil was slightly swollen and red. Under local treatment the tonsil became normal within five days. Careful inquiry in regard to previous diet and surroundings eliminated the possibility of scurvy.

The acute condition, the swelling of the right tonsil, the high percentage of lymphocytes in the peripheral circulation, suggested the possibility of acute leukæmia with an intercurrent infection from the right tonsil, giving rise to well-marked leukopenia. Other sources of infection, such as otitis media, hemorrhoids, pyorrhœa alveolaris, were carefully looked for and not found.

The decrease in leukocytes in leukæmia from 100,000 to 30,000 to 4000, or even lower in some of Cabot's cases, has occurred, so far as I am aware, only in cases with a severe intercurrent infection, such as pneumonia, empyema, septicæmia.

The evident absence of tonsillitis before admission, the sore throat occurring a few days later; the absence of pallor at any period preceding the first severe epistaxis; absence of glandular and splenic enlargement; the persistent bleeding from bowel, gums,

and nose; the purpuric eruption from the onset of the condition, suggests Werlhof's disease rather than acute leukæmia.

From the character of the lymphocytes found in the two blood examinations leukæmia seems excluded. Specimens from three cases of undoubted acute leukæmia examined in the laboratory show the large lymphocytes with "azur-granules" (erythrophilic, but not acidophilic)—Türk's lymphoid marrow cell[1]—in high percentage, and this type of cell was not found in this case, the mononuclear cells being of the lymphocyte and large mononuclear types found in normal blood.

A post-mortem examination was not permitted.

TWO CASES OF CHRONIC SPLENIC ANÆMIA (TWIN SISTERS), ONE WITH ACHYLIA GASTRICA.[2]

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The term splenic anæmia was first used by Griesinger to describe a condition characterized by a severe anæmia associated with chronic enlargement of the spleen. Since Gretsel reported the first case in 1866, an extensive literature has accumulated. An analysis of the cases leads to the conclusion that various diseases have been reported under this name. Some are undoubted cases of pernicious anæmia, others of pseudoleukæmia. In some it would be difficult to exclude malaria, syphilis, and tuberculosis. Banti was the first to make this a primary disease of the spleen. He believes that the enlargement of that organ precedes the anæmia, and that in the later stages the liver becomes cirrhotic; there is jaundice and ascites, eventually leading to death. But whether Banti's disease is a true splenic anæmia, or a cirrhosis of the liver beginning in the spleen, or whether the infantile pseudoleukæmia of von Jaksch belongs in this category, or whether primitive or primary splenomegaly, splenic pseudoleukæmia, Gaucher's endothelioma of the spleen, splenic cachexia, etc., and splenic anæmia are the same thing, or different stages of the same thing, are still mooted questions.

The term splenic anæmia is objectionable, as it assumes a causal relation between the splenic tumor and the anæmia. Objections,

[1] Cells resembling Türk's lymphoid marrow-cells, excepting the "azur-granulation" which Türk states he has not observed in these cells, although he suggests the possibility of the occurrence of such granules in the basophilic protoplasm of an "old" lymphoid marrow-cell. Türk, Vorlesungen über klinische Hämatologie, 1904, p. 366.

[2] Read at the annual meeting of the New York State Medical Society, January 31, 1905.

however, can be raised against every proposed substitute. In fact, the entire subject is yet in the stage of discussion. Wentworth, in a comprehensive review on the subject, concludes: "That the blood changes in the so-called splenic anæmia are those of a secondary nature." Also, "that it has not been confirmed that the splenic alterations are primary."

Rolleston, in a discussion on the pathology of splenic anæmia at the seventh annual meeting of the British Medical Association, said: "The fact that mistakes have been made, and from the difficulties presented by some cases will probably continue to be made, in assuming that splenic anæmia explains the morbid condition present in a given case does not prove that there is not a morbid entity, which, as it conforms to no other known type, must, at present at any rate, be tentatively recognized." Senator, during the same discussion, said: "I am of the opinion that so-called splenic anæmia cannot be strictly separated from similar affections of the hæmopoietic viscera which have a great resemblance to leukæmia without the characteristic condition of the leukæmic blood, and which, therefore, it is justifiable to class together under the name pseudoleukæmia, as Connheim proposed in 1865."

Osler, who has done much to give splenic anæmia its standing, and who has reported 18 cases, says: "A special malady does exist, of unknown etiology, characterized by a chronic course, enlargement of the spleen, anæmia of a secondary type, a marked tendency to hemorrhage of the stomach, and a liability in the late stages to be associated with jaundice, cirrhosis of the liver, and ascites." He also says: "It seems probable that the conditions separately described in the literature as primitive splenomegaly, splenic anæmia, splenomegalic cirrhosis of the liver, or Banti's disease are stages of one and the same malady."

It is my purpose to place on record 2 cases of chronic splenic anæmia occurring in twin sisters. The first case I did not see. I am indebted to Dr. Wallace for the notes. The second case was referred to me by Dr. Wallace. I have had the patient under observation since September.

Case I.—Mrs. J. H. H. when a child had frequent attacks of pain and enlargement of the joints; otherwise she was well up to her twenty-second year, when she was married. This was in 1892. She then weighed 128 pounds. Within six months her weight dropped to 105 pounds, and her general condition was poor. During 1893 she suffered from chronic diarrhœa and soreness in the left side. In 1895 she gave birth to twins. One child (male) was born dead, the other (female) lived three weeks. When two weeks old, rapidly growing, red, sessile tumors, as large as a hen's egg, formed on the posterior surface of the elbows. There was no rash or other abnormality.

In 1898, while at Grace Hospital, Toronto, where she expected

to have a lacerated perineum repaired, an enlargement of the spleen was discovered. Her general condition was poor, and an operation deemed inadvisable.

During 1899 she was anæmic. The ankles were enormously swollen.

Examination February 6, 1900, showed considerable improvement. Her weight was now 130 pounds. The lungs, heart, and pelvic organs were normal.

Abdomen large and distended by a mass which occupied the entire left side, extending below the crest of the ileum, and beyond the middle line. It was hard, smooth, slightly movable, and notched on the anterior border; the liver was not enlarged; there was no ascites.

Urine, cloudy, 1025, acid, large amount of albumin.

Blood, hæmoglobin, 75 per cent.; red corpuscles, 4,320,000; white cells, 850 per cubic mm.

In May, 1900, she had a severe attack of stomatitis. The gums were ulcerated; the submaxillary glands were painful and swollen.

The temperature ranged from 101° to 103°. There was no change in the abdominal findings. The urine was free from albumin.

Blood, hæmoglobin, 30 per cent.; red blood cells, 3,072,000; white cells, 1300. During July and August of that year her condition was wretched. She was emaciated, weak, and had a running temperature. Her death was expected. In the fall she began to improve, and in December she felt quite well and strong, and left the city.

On June 29, 1901, she was in the city for a day. Her condition was fair. Her skin was sallow, with irregular large brown spots. The tumor was about the same.

During the summer of 1902 she passed some renal calculi. In October she had another attack of stomatitis. The submaxillary and cervical glands were painful and enlarged. Her temperature ranged from 100° to 104°. There was some dulness at both apices. Sputum negative. From now on her condition grew worse.

Examination December 22d showed much emaciation. Blood, hæmoglobin, 35 per cent.; red blood corpuscles, 2,100,000; white blood cells, 1200. Differential count, polymorphonuclears, 35.5 mononuclears, 66.5. Sputum negative. She complained of pain in the lower part of the abdomen (right side). There was nausea. The breath was foul. Sputum and stools streaked with blood.

December 27th. During the afternoon she was suddenly taken with excruciating pain in the lower part of the abdomen. The abdomen was tender; pulse ranged from 100 to 140; temperature, 102°.

28th. Gradual failing of the cardiac and respiratory functions. Death occurred at 9.15 A.M.

Autopsy made by Dr. W. H. May at 11 A.M. Body much emaciated, rigor mortis present. There was a little dark yellow subcu-

taneous fat. The diaphragm reached to the third costal cartilage on both sides.

Chest. Pericardial sac contained about two ounces of fluid. The heart was apparently normal; weight four and one-half ounces. There was no fluid in the pleural cavities; the right lung was adherent at the apex and along the right border. The left lung was slightly adherent.

Abdomen. Full of pus below the mesocolon. No evidence of fat necrosis or venous thrombosis. Small intestine and cæcum injected, matted together, and covered with lymph. Mesenteric glands enlarged. The mucous surface of the alimentary tract apparently normal. Appendix, one and one-half inches long, thick, dark, inflamed, and adherent, but not perforated. Stomach free on both surfaces; no evidence of ulceration. The spleen occupied the entire left half of the abdomen, and was adherent to the diaphragm. It weighed a little over two pounds. There was no pus near it. Right kidney apparently normal; not dilated. It contained a calculus, about the size of an almond; also much sand. The ureter was about the size of the little finger, and about its middle there was a calculus as large as a bean. The left kidney was apparently normal. The suprarenals were small, dark, and friable. The liver was apparently normal. It weighed two and three-quarter pounds. The pancreas and pelvic organs were apparently normal.

Nothing was found to account for the pus, unless it was the appendix. It is safe to assume that it was the result of an acute process and had nothing to do with the chronic condition.

Dr. H. S. Steensland, Director of the Pathological Laboratory of the College of Medicine, Syracuse University, examined sections of the spleen, and reported a marked increase in the amount of reticulum, especially in the splenic pulp, but to a certain extent also in the lymph nodules. Endothelial cells filling the pulp spaces, as observed in the cases described by Gaucher, Bovaird, Harris, and Herzog, etc., were not present.

Case II.—Miss M., nurse, aged thirty-one years. Father died at sixty-two years of some stomach trouble. Mother is sixty-four years old, and well. Four brothers and three sisters are living and in good health. One sister died at thirty-eight years of peritonitis. One sister (twin) died at twenty-nine years (Case I. of this paper). In 1880 she had measles. From the fourteenth to her seventeenth year she was troubled with lumps or swellings on the hands, knees, and feet. After menstruation set in they almost entirely disappeared. In September, 1898, she had an attack of pleurisy, with effusion. In July, 1898, she had a severe attack of diarrhœa, which lasted about four weeks. At this time she also complained of soreness in the left hypochondriac region. The pain persisted about two months after the diarrhœa ceased. She then entered the training-school for nurses at the Hospital of the Good Shepherd. Hospital work agreed

with her. She gained in weight. Her color was good, and she was known as the rosy-cheeked nurse.

In January, 1899, she left the hospital to nurse her sister. In August of that year she had a second attack of soreness in the left side. Dr. W. L. Wallace examined her, but could find no apparent cause for it. The attack lasted some weeks and then disappeared.

For the next two years she was in indifferent health. At times she felt fairly well and strong; at other times she felt run-down and weak, and was forced to keep to her bed. In June, 1902, she again entered the hospital training-school. The work proved too much for her, and, after being on duty for two weeks, she was forced to turn patient, suffering from swelling of the left knee, left elbow, and right large toe. The diagnosis of inflammatory rheumatism was made. After two months' illness she was able to leave the hospital. Change of scene brought about a marked improvement in her condition. About the middle of September she again undertook to nurse her sister. This she did until the latter's death, December 28th. She endured considerable physical as well as mental strain. During 1903 her condition was poor. She was anæmic, had pains in the extremities, also in the left hypochondriac region. She had occasional attacks of diarrhœa. At other times she was constipated and suffered from sharp attacks of colic. She also had several attacks of stomatitis. The gums would become red and swollen. She also had a mild fever during the attacks.

In November, 1903, while visiting in Toronto, Canada, she consulted Dr. Cotton. He examined her, but could not determine any splenic enlargement. He found some thickening in the region of the appendix and advised an operation. This she declined, and returned to Syracuse. Her condition again improved.

In February, 1904, she had an attack of quinsy. The convalescence was very slow. The soreness in the side continued. A blood examination made by Dr. Wallace showed hæmoglobin 70 per cent.; red blood corpuscles, 3,680,000; white blood cells, 3600; differential count, polymorphonuclears, 17.5 per cent.; lymphocytes, 46 per cent.; large mononuclears, 32.5 per cent.; eosinophiles, 4 per cent.

In July she again visited Canada, and again consulted Dr. Cotton, and now for the first time he discovered that the spleen was enlarged, and advised splenectomy.

In October she returned to Syracuse and consulted Dr. Wallace, who kindly referred her to me.

Patient is thin, but not very anæmic. On the face there are a number of irregular brownish spots. The mucous membranes are of fairly good color. Tongue coated.

Lungs normal; heart not enlarged; sounds accentuated, but no murmurs. Pulse of moderate volume; some arrhythmia. The stomach does not reach below the navel. The liver dulness begins

at the fourth rib. The lower border is just palpable at the free border of the ribs, but is not enlarged.

The spleen is freely movable, and extends three fingers' breadth below the free border of the ribs. It is firm, smooth, and notched on its anterior border.

The right kidney is freely movable. The left kidney is also movable, but to a lesser degree. None of the glands are enlarged. There is some tenderness over the sternum; none over the other bones.

The blood examination shows a mild anæmia of the chlorotic type. Hæmoglobin, 80 per cent.; red blood cells, 4,800,000; white cells, 1600. The differential count shows polynuclear neutrophiles, 13 per cent.; lymphocytes, 46 per cent.; large mononuclears, 40 per cent.; eosinophiles, 1 per cent. No poikilocytosis; no nucleated reds, but some inequality in the size of the erythrocytes.

A blood count, made October 15th, resulted: Hæmoglobin, 70 per cent.; red blood corpuscles, 4,700,000; white cells, 600. There were so few white cells that it was difficult to make a differential count. It was, however, very evident that the mononuclear cells far outnumbered the polymorphonuclears.

On account of the frequent attacks of diarrhœa, I determined to make an analysis of the stomach contents. She was given the ordinary Ewald test-breakfast, consisting of 300 c.c. of water and 50 c.c. of bread, and the contents aspirated one hour later. The obtained contents formed a thick, gelatinous mass, consisting of much mucus mixed with the food particles. The bread was not well digested. A chemical examination revealed that free hydrochloric acid was entirely absent, the total acidity was 4. There was neither pepsin nor rennet present. Two days later I gave her a test-meal consisting of soup, meat, bread, and vegetables. Four hours afterward the contents were aspirated. The thick, gelatinous mass passed through the tube with difficulty. Macroscopically the various ingredients of the meal could be distinguished. An analysis gave the same result as before. No free hydrochloric acid, no pepsin, no rennet, and a small amount of combined acids.

Since then I have made repeated examinations of the stomach contents, at various times of the day and under varying conditions, and each time with the same result—a complete absence of free hydrochloric acid and the ferments; in other words, a true achylia gastrica.

Repeated blood examinations were also made. The result was always about the same. The red cells varied from 4,100,000 to 4,800,000, the whites from 600 to 2700. The differential counts, likewise, gave similar results. The polynuclear neutrophiles were always in the minority. The highest polynuclear count was 30 per cent.; the lowest 11 per cent. At no time were there any myelocytes or nucleated reds present, nor was there any poikilocytosis. In estimating the white cells the Türk counter was used, the entire chamber,

equivalent to nine ordinary Thoma slides, being counted. In the differential counts I followed Türk's classification of the white cells. The transitionals were counted with the large mononuclears, while under lymphocytes, both large and small lymphocytes were included.

These cases present some interesting features. First, the presence of the disease in twin sisters. Bovaird, Brill, Collier, Wilson, Springthrope, and others have reported instances of splenic anæmia occurring in more than one member of a family. Whether this is merely a coincidence or the result of an infection or of some congenital predisposition cannot be positively asserted. Little is known of the etiology of the disease. Micro-organisms have been searched for, but no specific germ has been isolated. The disease resembles a toxæmia. Whether the toxin is produced in the spleen or gastrointestinal tract cannot in the present state of our knowledge be answered. The fact that improvement and even cure have followed splenectomy would indicate that the spleen plays a responsible part. However, it is generally believed that the gastrointestinal tract is the source of the poison.

The marked leukopenia is also noteworthy. In Case II., one count showed but 600 white corpuscles per cubic millimetre. Here, also, the question might be raised; are we dealing with a process of cell destruction or with a disturbance of cell production? We know that hæmolysins and leukotoxins are produced in the blood. But we are not familiar with the production of auto-hæmolysins or autoleukotoxins. The differential counts in both cases were a little unusual. Ordinarily, in splenic anæmia the differential count shows nothing characteristic. In both of these cases there was a marked relative as well as absolute decrease of the polymorphonuclear cells, and a decided relative increase of the large mononuclear leukocytes.

Another interesting feature is the stomach findings in Case II. We have the condition known as achylia gastrica (Einhorn). The continuous absence of free hydrochloric acid and the ferments in the gastric contents. Achylia gastrica is frequently associated with pernicious anæmia. In fact, the absence of free hydrochloric acid is more frequently associated with this disease than with any other, not excepting cancer of the stomach. The relation of the stomach mucosa to the blood condition has been much discussed. It is safe to say that both are due to the common toxic agent and do not bear the relation of cause and effect to each other. A short time ago Nothnagel called attention to atrophy of the intestinal mucosa as the cause of pernicious anæmia. Faber has recently investigated this question. He concludes that in pernicious anæmia there is no change either in thickness or the character of the intestinal mucosa. He ascribes Nothnagel's findings to post-mortem changes. Whether achylia gastrica is only a coincidence in this case, or whether it is as common with splenic anæmia as with pernicious anæmia, the examination of the stomach contents in other cases will show.

Both cases also show a marked tendency to periods of remission, during which there is a decided improvement of the symptoms. Such periods of remission are quite common in the diseases of the blood-producing organs, like pernicious anæmia, leukæmia, and pseudoleukæmia.

The effect of arsenic in Case II. was rather striking. The patient had at various times taken the drug—each time with a marked aggravation of the symptoms. The lowest white count was encountered while she was taking Fowler's solution. This corresponds with the known physiological action of arsenic, namely, that it stimulates the production of the erythrocytes, while it inhibits the formation of the white cells. Walter Broadbent has called attention to the dangers of the indiscriminate use of arsenic. He believes that many symptoms, like pigmentation of the skin, œdema, ascites, and the sensory disturbances of pseudoleukæmia, leukæmia, and pernicious anæmia are due to the arsenic taken.

The question of operation must be considered in these cases. In some cases splenectomy has brought about a cure; in others it has prolonged life. Knowing the powerlessness of drugs, the knife may be appealed to in suitable cases, even though its use is purely empirical.

A CLINICAL AND PATHOLOGICAL REPORT OF A CASE OF SPLENIC ANÆMIA.

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AND

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1. Clinical Report (Dr. Sanford). *Case of anæmia of long duration with gastrointestinal disturbances and pain in splenic region; recent severe and repeated hemorrhages by stool and vomitus following traumata; blood characteristic of secondary anæmia of chlorotic type without leukocytosis, with no general lymphatic enlargement; idiopathic splenomegaly; exploratory laparotomy; subsequent splenectomy; death.* Mr. B. R. T., aged twenty-eight years, married, a farmer, of Greenwich, Ohio, was sent to Lakeside Hospital on the evening of October 14, 1903, with symptoms of vomiting of blood, tarry stools, profound anæmia and a tumor in the lower mid-abdomen.

The patient stated that three weeks before he had been thrown over the head of a spirited horse and had struck flat on his back, but had not been prevented from working about as usual. Two

weeks later, which was a week before entrance, he was jerked off his feet and thrown violently some distance by this same horse which he was leading. The following morning he had a copious black liquid stool, after which he vomited much blood, he thinks at least a pint, of both dark blood clots and some fresh blood. This vomiting continued at intervals that day, and then stopped, but he had large tarry stools nearly every day up to the time of his entrance. He was in bed during most of the time, felt greatly prostrated, and had no appetite.

His physician on the day before he was brought to the hospital first noticed a tumor in the lower mid-abdomen, which he took to be a distended bladder, but on catheterization its size was not altered. The urine appeared normal and contained no blood. Dr. Maynard, of Elyria, who was called in consultation, then sent him to the hospital. On entrance the patient seemed greatly fatigued from his journey and could answer questions with difficulty.

Physical Examination. Patient is a young American, slight in frame, very sallow, somewhat emaciated, in considerable state of shock. Temperature, 100.6°; pulse, 120, small volume, regular, compressible; respiration, 28.

Sclera subicteric; teeth poor; tongue furred and moist.

Chest, slender and shallow, expansion equal and good; clavicles and ribs prominent; supraclavicular and infraclavicular fossæ deep; no glands palpable. Interspaces sunken; epigastric angle narrow.

Percussion note dull over clavicles and supraclavicular spaces, elsewhere hyperresonant. Breath sounds clear front and back; musical rales on expiration in left foreaxillary line.

The area of cardiac dulness is hard to define; it apparently extends somewhat to the right of the sternum; a blowing systolic murmur is heard all over the base, and not at the apex or in the axilla.

The abdomen is sunken, scaphoid in general outline, somewhat sensitive to palpation in the left upper quadrant where there is slight muscular rigidity. Liver dulness begins at the fifth space in nipple line and extends to costal margin. Splenic dulness not made out. The pole of right kidney is palpable on deep inspiration. Left kidney not felt, and it seems with one hand in the left lumbar region and the other under the left costal margin that the two hands can be brought nearer together with less intervening structures on the left than on the right side. On pressure here the patient is evidently sensitive, although apathetic to examination elsewhere. Just below the umbilicus is a mass, oval in general outline, lying diagonally, its higher pole to the right, smooth, hard, not tender, quite freely movable, edges rounded, no notches felt. The abdomen is everywhere tympanitic except over the mass described where the note is flat.

The inguinal glands are slightly enlarged. Rectum empty except for a few small clots; no hemorrhoids; extremities thin; no tibial scars or œdema.

The patient was given artificial heat and stimulation by strychnine and saline subcutaneous infusions every six hours. Milk and whites of eggs; morphine for restlessness. A glycerin enema resulted in a black liquid stool, which on examination was found to be largely decomposed blood.

The immediate history of severe repeated traumatisms combined with the patient's statement that he had always been well previously, quite naturally led one to regard the present symptoms as the direct result of the injury rather than to seek a more remote cause. Various possibilities were considered, among which hemorrhage from a gastric or duodenal ulcer, and a dislocated viscus were foremost. Neither diagnosis seemed to account for all the symptoms, and in view of the later developments in the case, a further discussion of these points is unnecessary.

The patient reacted fairly well to stimulation, and was kept under observation the next day, during which he had seven more black liquid stools. Rectal feeding was added to the treatment which was otherwise the same.

A blood examination on the day after admission showed: Reds, 2,800,000; leukocytes, 8000; hæmoglobin, 25 per cent.; coagulation time 45 seconds; no plasmodia seen.

The next day the patient's unchanged condition and the uncertain diagnosis seemed to warrant an exploratory operation which was decided upon.

Operation, October 16th (Dr. Dudley P. Allen. Gas and ether). *Laparotomy; exploration.* An incision 9 cm. in length was made in the median line below the umbilicus and over the tumor. A small quantity of clear peritoneal fluid was met with from which cultures were taken. The intestines were normal. The mass was identified as the spleen, greatly enlarged and displaced, freely movable, otherwise of normal appearance. Accurate measurements were not taken, but the organ seemed at least two or three times its normal size. The surgeon considered any operative procedure on the spleen unwise in the patient's condition and closed the incision. Another incision in the median line of the epigastrium was then made and the stomach inspected, and found normal. Nothing abnormal felt about the liver, gall-bladder, appendix, or pancreas. Palpation of the kidneys showed them apparently normal in position and size. The incisions were then closed. The patient received very little shock from the operation, which took but fifteen minutes.

The conditions found at operation raised the question whether the traumata might not be coincidences or, perhaps, exciting causes in producing the appearance of symptoms primarily due to some

as yet unrecognized organic lesion. The arrival of the patient's friends gave the first opportunity of getting the family and previous history in the case, which was as follows:

Family History. Father died of "broken back" at the age of seventy years. Mother and one brother living and well. An uncle died of heart disease. No history of malaria, syphilis, cancer or tuberculosis in the family.

Previous History. Patient had children's diseases. He has always been pale since childhood. Between seven and eight years ago he had an attack of very violent vomiting which continued all night and was not connected with any indiscretion in diet; vomitus contained no blood. He has always had poor digestion, often distressed after meals, sometimes vomiting, with relief to the discomfort.

In the summer of 1902 he had "bowel trouble," in which there was diarrhœa, griping pains, severe vomiting, with some blood in stools and vomitus. From this time up to the injury the patient was not well, and was under a doctor's care for general "ill feeling" and frequent urination. In the winter of 1902, he also complained of a good deal of dragging pain in the back and left side. No history of venereal disease; tobacco in moderation; no alcohol. Patient has not lived in a malarial district and never had periodic chills.

With this suggestive history and the enlarged and dislocated spleen found at operation, a detailed blood examination was then made and showed the following conditions: Reds, 3,480,000; leukocytes, 8500; hæmoglobin, 25 per cent. Differential count of 600 cells: polymorphonuclears, 74 per cent.; small mononuclears, 18 per cent.; large mononuclears, 4 per cent.; transitional forms, 1.5 per cent.; eosinophiles, 2.5 per cent. Red cells took stain poorly, and were variable in shape and size, centre quite thin, no rouleau formation, some poikilocytosis, six nucleated reds seen; no plasmodia.

The urine was amber, clear, specific gravity, 1020, acid, no sugar, faint trace of albumin, sediment, few leukocytes, epithelial cells.

The case then presented the following data: A patient with (1) a previous history of an anæmic condition with gastrointestinal disturbances and pain in the splenic region; (2) an immediate history of severe and repeated hemorrhages by stool and vomitus following traumatism; (3) a present condition of profound anæmia, with the blood characteristic of a secondary anæmia of the chlorotic type without leukocytosis, with no general lymphatic enlargement; and (4) a splenomegaly apparently not due to malaria, syphilis, tuberculosis, leukæmia, or amyloid disease. From this symptom-complex the possibility of the existence of splenic anæmia was strongly suggested and a course of supporting treatment was instituted while further study could be carried on.

During the week after operation the patient's general condition improved slightly. The stools no longer contained fresh blood,

but were dark, copious, watery, varied between three and seven daily and were at times involuntary. There was no further vomiting. Liquid nourishment was well taken. The temperature ranged between 99.5° and 101.5°, and the pulse from 100 to 140. The outline of the tumor was marked out on the abdomen in silver nitrate solution to watch for changes in its size or position.

On the tenth day after operation the tumor was found to have increased in size by almost one-half, and the gain in size seemed mostly toward the right and below. The new outlines of the enlarged organ were marked on the patient's abdomen. Two days later a still further very rapid increase in size was apparent, so that the anterior surface of the mass presented an area twice as large as when the patient entered the hospital. The incisions of the original exploration were firmly healed by first intention. To-day for the first time petechial hemorrhages were seen on the sides of the chest in areas the size of the palm of the hand, and smaller similar purpuric spots appeared on the right forearm and hand.

No change in subjective symptoms accompanied these new phenomena. The patient was still apathetic and weak, though he declared he felt better, and his only complaint was the exhaustion attendant on frequent bowel movements which still were of a tarry color and averaged five to six daily. The blood condition was examined from day to day, and showed practically the same condition as given above.

The question of splenectomy in the case had been constantly before the surgeon's mind, but he desired first to improve the patient's general condition in view of the recent enormous loss of blood he had suffered. The rapid and sudden increase in the size of the spleen, however, convinced Dr. Allen that ground was being lost instead of gained, and an immediate operation was proposed and accepted by the patient and his friends.

Operation October 29th (Dr. Dudley P. Allen. Gas and ether). *Laparotomy; splenectomy; closure.* A vertical incision 15 cm. in length was made separating the fibres of the left rectus muscle in order to avoid the former incisions. On opening the peritoneum some clear fluid was met with from which cultures were taken. The spleen was found to be lying free in the abdomen without adhesions to the intestines or other structures, its only attachment being a large long pedicle containing four twists, composed of large congested vessels, some of which contained thrombi. A more detailed, microscopic description of the organ and its pedicle will be given later.

The pedicle was securely tied high up with silk, clamped off distally and removed with the organ, which was with difficulty squeezed through the skin incision. No hemorrhage accompanied the removal; the pedicle stump was reinforced by over-and-over catgut stitches, and showed the openings of several large vessels.

The abdomen was closed in layers. The patient seemed to receive no shock from the operation which lasted twenty minutes.

For two days after operation the patient's general condition was encouraging. The temperature approached the normal, and the pulse rate decreased to an average of 90, with improvement in its quality. Stimulation with strychnine and saline subcutaneous infusions was continued as before.

On the third day a marked œdema of the whole right leg appeared which increased rapidly to alarming proportions. This was followed some days later by œdema of the right side of the scrotum and left side of the neck. Vomiting reappeared on the sixth day, and it became difficult to nourish the patient, as rectal nutrition was not retained except at intervals. There was no further appearance of blood, however, either in the vomitus or stools. Purpuric areas remained unchanged. Later the vomiting ceased and the patient seemed much brighter again. An attempt was made to give Fowler's solution, but this had to be abandoned, as on the eleventh day the patient again rejected all nourishment. The abdomen, which up to this time had been flat, now began to show distention, with appearance of free fluid in the flanks. This collected rapidly, and on the thirteenth day the abdominal wound, which had healed and from which the stitches had been removed, burst open and a large amount of clear fluid escaped, from which cultures were sterile.

The patient now was irritable and very hard to rouse, sleeping nearly all the time with some delirium. Nourishment was refused, and rejected when given. Stools and urination were involuntary. This condition of progressive weakness, accompanied by emaciation, dyspnœa, and cyanosis, continued until the patient's death on November 15th, seventeen days after operation.

The changes in the blood following the removal of the spleen were very interesting, and in the main coincided with observations reported under like conditions in other cases, though, unfortunately, the patient's death prevented their being followed out over a long period.

Immediately after operation there was a great diminution in the red corpuscles from 3,480,000, four days before operation, to 1,836,000 two days after operation. This could not be accounted for by hemorrhage, either before or during the operation, as there was no great loss of blood immediately before the operation and no blood was lost in tying off the pedicle of the spleen. It must be attributed to the effect of the removal of the organ. After this initial drop the red count began steadily rising and had reached 2,800,000 just before death.

The white count which had been 8500, jumped to 34,000 two days after operation and gradually decreased to 22,000 at death. The percentage of hæmoglobin which before operation was 25 per cent., dropped to 22 per cent. after operation, and then gradually rose to

28 per cent. All these changes, the marked postoperative diminution in red cells, and to a less degree in hæmoglobin, and the sudden rise in leukocytes, corroborate previous experiences following splenectomy.

In the differential counts the polymorphonuclear form showed a marked increase from 74 per cent. before operation to 93 per cent. before death. The eosinophiles varied in amount. Nucleated red forms which averaged 6 in a count of 600 cells before operation increased to 36 before death.

To facilitate comparison the results of the various counts are tabulated below.

	Date.	Red cells.	Leukocytes.	Hæmoglobin.
Before operation	October 16	2,800,000	8,000	25 per cent.
	" 25	3,480,000	8,500	25 "
After splenectomy	November 2	1,836,000	34,800	22 "
	" 3	1,914,000	30,000	23 "
	" 7	2,212,000	22,000	22 "
	" 10	2,848,000	23,720	28 "
	" 11	2,464,000	26,000	26 "
	" 12	2,568,000	23,000	27 "
	" 13	2,632,000	22,000	28 "
	" 14	2,682,000	22,000	28 "

DIFFERENTIAL COUNTS OF 600 CELLS.

	Date.	Polymorphonuclear.	Small mononuclear.	Large mononuclear.	Transitional.	Eosinophiles.
Before operation	Oct. 25	74.0 per ct.	18.0 per ct.	4.0 per ct.	1.5 per ct.	2.5 per ct.
After splenectomy	Nov. 2	83.5 "	12.75 "	1.75 "	1.50 "	0.5 "
	" 7	89.0 "	4.66 "	2.33 "	2.0 "	2.0 "
	" 10	93.0 "	3.8 "	1.4 "	0.6 "	1.2 "
	" 13	92.3 "	3.6 "	1.3 "	1.0 "	1.8 "

2. PATHOLOGY. (Dr. Dolley.) *Autopsy Protocol.* (Autopsy performed seventeen hours after death.) The body is that of a well-formed but emaciated white man, 180 cm. long. There is moderate rigor mortis, and slight posterior hypostasis. The conjunctivæ are somewhat yellow. The mucous membranes are pale. The skin is sallow but not pigmented. A few small petechial areas are scattered just above the costal border. There is no glandular enlargement.

The chest is barrel-shaped. The supraclavicular fossæ and sternal notch are deep. The abdomen is scaphoid. Two recently

healed wounds of operation appear in the midline; one, 6 cm. long, beginning at the xiphoid cartilage, the other, 7 cm. long, beginning at the umbilicus. A third laparotomy wound, healed with the exception of one stitch, 12 cm. long and 3 cm. to the left of the umbilicus, is present in mid-abdomen. The right leg is larger than the left throughout, and moderately œdematous.

Brain and *spinal cord* not examined.

Abdominal Cavity. The spleen is absent. The splenic veins become very tortuous from the end of the pancreas, one and one-half coils being left after the splenectomy. They measure from the end of the pancreas to their ligated extremity 2.5 cm. coiled and 5 cm. uncoiled. The apposing surfaces of the coils are united by well-organized adhesions. Beginning at the junction with the portal vein, the splenic vein and its branches become more and more distended with palpably soft material until at their ligated extremity the larger of the two branches is 2 cm. in diameter. The veins are attached to the jejunum at one spot, 2 cm. in diameter, by organizing fibrinous exudate. On incision they contain soft mixed thrombi, which in places are loosely attached to the wall. There is slight thickening of the walls but no evidence of calcification.

The *splenic artery* is rather tortuous and dilated. It measures 2.5 cm. from the end of the pancreas to its ligated extremity. Its elasticity is poor, the intima is somewhat roughened and extremely reddened toward the distal end. It contains a similar grayish-red thrombus.

Mediastinal fat is scanty. The *thymus* is absent.

Pleural Cavities. Each contains about 10 c.c. of clear serous fluid. There are no adhesions.

The *pericardial cavities* contain 30 c.c. of similar fluid. The sac wall is not thickened.

Thyroid Gland. Both lobes are somewhat enlarged. The tissue appears normal.

The *tracheal glands* are markedly anthracosed. One at the bifurcation is studded with small fibrocaseous tubercles.

The *lungs* are voluminous, externally smooth and slightly anthracosed. They are hypercrepitant and cushiony except in the lower posterior portions. On section there is moderate hypostasis of the lower lobes. Elsewhere the tissue is more moist than normal. The left weighs 550 grams, the right 549 grams.

The *bronchi* contain some frothy watery mucus.

The *bronchial glands* are like the tracheal. One is composed of a calcified envelope with a caseous centre.

The *heart* is of normal size. The right ventricle averages 4 mm., the left 15 mm. in thickness. The auricles and right ventricle contain post-mortem jelly clots. Fluid blood is scant. The muscle is pale and flabby. The valves are normal except for a few atheromatous patches on the ventricular surface of the right mitral

segment. The circumferences of the valvular orifices are: aortic, 6.2 cm.; pulmonic, 7.3 cm.; mitral, 9.5 cm., and tricuspid, 13 cm. The coronary arteries have a roughened intima with considerable atheroma. They are not tortuous. The endocardium elsewhere is smooth.

Aorta. The elasticity is normal. The wall is not thickened. The intima in places is roughened by yellowish or whitish plaques.

The *pulmonary artery* and *venæ cavæ* appear normal.

The *liver* weighs 1356 grams. It measures 27 x 14 x 8½ cm. The left lobe measures 13½ x 14 x 5 cm. It is contracted, but its shape is fairly preserved. The capsule is slightly thickened, and the capsular veins are moderately injected. The veins of the round ligament are not dilated. The surface is moderately roughened and nodular, especially over the inferior and lateral portions of the right lobe. Over the inferior surface are several nodules softer in consistence than the rest of the tissue. On section, the organ is moderately hyperæmic. The cut surface is uneven and finely, though not uniformly, granular. The tissue is firm and cuts with increased resistance, especially in the lower and lateral portions of the right lobe, just under the capsule. The lobular outlines are not distinct; the tissue is markedly bile-stained, of a dirty brownish-yellow color. Owing to the bile staining the character of the apparently increased connective tissue is not evident. Glisson's capsule is most affected.

The *portal vein* is not thickened and its intima is smooth; it is free from thrombi.

The *gall-bladder* is constricted at the junction of its proximal and middle thirds and bent upon itself, so that the two portions are in partial apposition and united by old adhesions. Elsewhere it is non-adherent and apparently normal, containing dark bile. The ducts are patent.

Œsophagus. Congested sub-mucosal veins are distinct about the cardiac orifice.

The *œsophageal veins* are considerably dilated and tortuous. The coronary veins of the stomach are not so dilated, but the cardiac branches communicating with the œsophageal veins show marked varicosity.

The *stomach* is somewhat dilated, but the wall is not appreciably thinned. It contains 500 c.c. of brown sour-smelling fluid. The mucosa shows considerable post-mortem change and is not indurated. The sub-mucosal veins about the cardia and along the greater curvature are dilated. The folds of the mucosa only appear along the greater curvature, where their prominence is due to dilated veins. No eroded veins are apparent. There are no ulcers nor scars.

The *pancreas* is moderately hyperæmic. The head and body are of normal size, but are distinctly indurated. The tail is very con-

tracted and firmly attached to the splenic vessels. On section there is great increase of dense fibrous tissue.

The *small intestine* is not distended. It shows only an occasional congested but not swollen area in the mucosa. The veins of the serosa are not congested.

The *appendix* is normal.

Large Intestine. The mucosa of the cæcum and the ascending colon is markedly congested. There are no hemorrhoids.

The *suprarenals* show post-mortem softening of the medulla.

Kidneys. Right weighs 215 grams, the left 285 grams. They measure respectively 12 x 7.5 x 3.8 and 13 x 8.6 x 7 cm. Perirenal fat is scanty. The cortical veins are not dilated. The capsule is normal.

The cortex of the right is 6 mm., of the left 8 mm. in thickness. The cut surface is smooth, but mottled with grayish areas corresponding to the labyrinths. The organs are moderately pale and the glomeruli are barely visible. The arteries are not sclerotic.

The *ureters, bladder, seminal vesicles, prostate,* and *testicles* appear normal.

The *mesenteric glands* are small and barely palpable.

The *retroperitoneal glands* are enlarged, firm, and of a glistening grayish-white appearance on section.

Hæmolymph Glands. Lying on both sides of the sternum, along the carotid arteries, the renal vessels, the iliac vessels, and about the splenic vein and the œsophagus appear glands varying in size from a pinhead up to that of a large bean. These are soft, dark-red, spleen-like and fairly abundant. Between thirty and forty of convenient size were removed.

The *right iliac vein* just above Poupart's ligament is filled with a red, non-adherent thrombus.

The *right femoral vein* contains a similar thrombus. This changes in character 5 cm. below Poupart's ligament, having a white friable core which at one point is attached to the wall.

The *inferior hemorrhoidal veins* are moderately dilated and tortuous.

Bones. Several ribs and the right femur were opened and found to contain bright-red and succulent marrow. No lymphoid areas appear.

Spleen (received from the surgical department. Gross description by Dr. Howard). The specimen consists of a spleen, irregularly oval in shape, presenting a rounded anterior margin from which the notch has been obliterated. It weighs 1650 grams and measures 22 x 16 x 7 cm. The vessels, which form a pedicle, are covered with fine pinpoint fibrin masses, but present no adhesions. This pedicle is twisted four times, and its uncoiled state measures 17 cm. in length and 4 cm. in its greatest diameter. It is composed of several large tortuous veins surrounded by a considerable amount of fat,

and several branches of the splenic artery, which is divided a considerable distance from the hilus. The pedicle also includes a soft body measuring 1.5 x 1 cm., covered with peritoneum, and resembling an enlarged lymphatic gland. At the distal end mixed gray and red thrombi protrude from the veins. The largest vein is 2.5 cm. in its greatest diameter. On incision they are filled with thrombi, but contain a variable amount of thin fluid blood. The thrombi are partly red and partly mixed, some being yellowish-gray in color. As a rule they are non-adherent, or can be easily separated. In general the mixed thrombi are soft or friable. The thrombi can be readily traced into the large and the small veins of the organ. The walls of the veins are not much thickened, and show no signs of recent or old inflammation except the thickening of one near the hilus.

The splenic tissue is light brown in color and œdematous. The trabeculæ are well marked, the lymph nodes obscure. The tissue is not friable. There are no infarcts.

Microscopic. Material from all organs was hardened in Zenker's and Orth's fluid, in formalin and in alcohol, and stained by ordinary and special methods.

Lungs. There is moderate emphysema and anthracosis. The sections from the posterior portions show moderate congestive œdema. The bronchi and pulmonary arteries are normal. Careful search for the bone-marrow giant cells described by Warthin in his cases was negative.

Heart. There is marked segmentation and some fragmentation of the muscle.

Liver. The connective tissue is moderately increased, but not at all uniformly even in the same section. The increase is greatest in the right lobe under the capsule, as noted macroscopically, Glisson's capsule about the larger vessels being particularly thickened. The lobules are usually sharply defined, but occasionally the connective tissue extends somewhat into them. The connective tissue is dense fibrous in character, with few nuclei, and slight round cell infiltration. In places of more marked fibrosis, fibroblastic cells are more abundant, but the tissue is nowhere actively proliferating. More rarely an increase of fibrous tissue about the central veins is noted. Mallory's connective-tissue stain shows here and there a slight increase of the reticulum of the lobules. This is more prominent in areas of atrophy of the lobular centres. This reticulum takes a light red with Van Gieson's stain. A very few fibroblastic cells appear in these areas and several capillaries are filled with proliferating endothelial cells with a few karyokinetic figures. Excepting these few instances endothelial proliferation does not occur. In the more cirrhotic portions many lobules show dilatation of the central veins and capillaries with some atrophy of the liver cells, but this is appreciable in but few lobules in the less fibrosed

areas. The atrophic liver cells present more or less fatty degeneration and necrosis, with a little brownish-yellow pigment. Numerous attempts to demonstrate the presence of iron were unsuccessful. Proliferation of bile canaliculi is found in only two areas. No bone-marrow giant cells are found.

The *stomach* and *intestines* show chronic passive congestion, associated in the small intestine with slight chronic catarrhal inflammation.

Pancreas. Sections from the head and body show a moderate increase of connective tissue, both perilobular and interacinous. Some islands of Langerhans also have an increased amount of stroma. The sections from the tail show marked fibrosis, dense fibrous and hyaline tissue replacing many lobules, while the remaining ones are small and distorted. The ducts have a thickened wall and a few are tortuous. The islands of Langerhans are diminished in number. A few are unaffected. In some there is fibrosis, but in others fatty and granular degeneration of the cells is more pronounced. The veins are moderately congested and have thickened walls, particularly in the tail.

The *kidneys* show a very slight chronic interstitial nephritis. There is no deposition of pigment.

The *portal* vein is normal.

Splenic Veins. The intima is slightly and irregularly thickened and is composed of dense fibrous tissue with few nuclei. Thickening of the adventitia is more pronounced. In the latter, particularly about the vasa vasorum, there is moderate fibroblastic proliferation. But few fibroblastic cells occur in the intima and media. Organization of the thrombi is well advanced in places.

The *mesenteric glands* are fibrosed. There is some proliferation of the endothelium.

Hæmolymph Glands. A few are of the marrow lymph type described by Warthin, but show partial transformation into ordinary lymph glands. The lymph spaces are full of endothelial cells, some of which are phagocytic for red blood and lymphoid cells. Only a moderate number of bone-marrow giant cells appear.

The majority of the hæmolymph glands resemble splenic tissue in structure. The follicles contain no germinal centres and usually show small hyaline areas. The blood sinuses, which are much dilated, are only partly filled with blood. The most striking feature is the presence within them of numerous large cells of endothelial type packed with red blood cells. Mononuclear eosinophiles appear in great numbers, and normoblasts are moderately numerous. A few mastzellen are present. Numerous cells resembling myelocytes appear in the blood sinuses, but these are more abundant in the lymph spaces of the glands of marrow lymph type (Wright stain). Pigment is in moderate amount. It gives the iron reaction with

ferrocyanide and HCl. Some phagocytic cells give the same reaction in a diffuse way.

The *bone-marrow* (rib) is of a poor lymphoid type. Giant cells are not apparently reduced in number. Plasma cells and polymorphonuclear leukocytes are in small proportion, but mononuclear eosinophiles with nuclei varying greatly in size are extremely abundant. Polymorphonuclear eosinophiles, while absolutely increased, are less numerous relatively. Normoblasts appear in large numbers with active mitosis. Myeloblasts are only occasionally seen. Neutrophilic myelocytes are almost entirely absent. A peculiar feature with the Wright stain is the presence of cells resembling lymphocytes, but with a red protoplasmic border. Excluding these, ordinary lymphoid cells are not increased in number.

There is a scanty amount of iron containing pigment, both in cells and free.

Spleen. The capsule is moderately thickened and composed of a dense hyaline connective tissue. The trabeculæ are also thickened and are farther apart than normal. The splenic tissue is constructed of considerably dilated blood spaces, more or less filled with blood, and separated by a moderate amount of usually dense fibrous tissue with a varying number of fibroblasts. Under the capsule this newly formed connective tissue is much more abundant, and it is not uniform in the other sections, thick fibrous bands appearing here and there. Hyaline transformation of the connective tissue appears nowhere outside of the trabeculæ proper. The venous spaces are lined either by an almost flat or a somewhat swollen endothelium with an occasional free cell, but no proliferating endothelium appears. Scattered through the pulp are relatively few lymphoid cells and an almost equal number of polymorphonuclear leukocytes. The lymph nodes are farther apart than normal, but it is not possible to say they are absolutely diminished in number. They are usually not atrophic but show an increase of coarse reticulum with a few fibroblasts. Often the central arteriole is thickened and hyaline. The veins are greatly dilated, their walls are moderately thickened and many contain fibrinous and mixed thrombi. Not infrequently small fibrinous clumps appear in the blood spaces.

A very moderate amount of pigment is deposited in phagocytic cells in the blood spaces or in the stroma or lies free in the trabeculæ. It gives the iron reaction. A few normoblasts, plasma cells, and mononuclear eosinophiles are to be seen, but neither myelocytes nor bone-marrow giant cells are found.

With the Van Gieson stain there is considerable variation, but a large part of the newly formed stroma takes a deep-red color. A section throught one of the splenic veins removed at operation shows the same degree of fibrous thickening as in the stump. The

adventitia is the seat of a much more marked fibroblastic proliferation. The thrombus is just beginning to organize.

Pathological Summary. There is nothing in the histological structure of the spleen which cannot be explained simply by chronic passive congestion. In the causation of this, two factors have to be considered—*i. e.*, the abnormality of the splenic vessels and the cirrhosis of the liver. The dislocation of the spleen must have been of long standing, for the splenic vessels uncoiled measured 19.5 cm. from the end of the pancreas. Granting even that the multiple twists all occurred as a result of the accidents, which is hardly probable, the elongation which existed prior to this must have caused considerable interference with the splenic circulation.

In two cases of splenic anæmia, reported by Dock and Warthin, there were stenosis and calcification of the portal vein, a condition identical with this one as far as the effect on the spleen is concerned. The question they raise is whether the portal lesion is primary and the splenic fibrosis secondary to it, or, on the other hand, whether the splenic condition is primary, while the condition of the portal vein is coincidental or results from a toxic condition of the portal blood dependent upon the disturbed splenic function or dependent upon a portal or general intoxication. The dislocation of the spleen could not have been of congenital origin, for the alteration does not indicate such a long-standing condition, and an enlargement of the organ at once suggests itself as the main factor in its production.

The abnormality of the splenic vessels is essentially a mechanical, not an organic one. Granted that some enlargement of the spleen, from whatever cause, started the elongation of the splenic vessels, once produced, with probably some torsion, the reaction on the circulation of the dislocated organ would be so great that the subsequently increased congestion and fibrosis would result largely from the condition of the vessels. The condition of the spleen must be considered then as largely secondary to that of the vessels, which gives them a very probable etiological significance. If splenic anæmia existed before the dislocation of the spleen, and the initial enlargement and the dislocation of that organ were manifestations of the pathological processes of the disease, the splenic vessels lose their casual relationship. But the histological structure offers no evidence of this. There is not even the proliferation of endothelium so commonly found in the spleen in this disease to complicate the picture.

The relationship of the cirrhosis of the liver to the condition in the spleen presents the greatest difficulty in interpretation. In part the process in the liver is regarded as a mild and not uniform chronic passive congestion, with a just beginning central cirrhosis, shown by the increase of fine reticulum in the atrophic portions of the lobules, with some endothelial proliferation. The moderate emphysema of the lungs offers a probable explanation for this

condition. More important there is also a moderate though irregular increase of periportal tissue, but the histological pictures of the fibrosis in the liver and in the spleen are too nearly identical to draw conclusions regarding time relationship. What part the liver played in the production of the splenic condition it is impossible to say. Was the hepatic process primary, and was the initial enlargement of the spleen leading to its dislocation secondary to it, or were the two processes coincident? The fibrosis in the liver and in part that in the spleen may have resulted from a general or portal intoxication.

The changes in the hæmolymph nodes are interpreted as compensatory for the failing splenic function. It is probable that the bone-marrow shared to some degree in the assumption of this function. But the presence of pigment and phagocytes in the spleen would, however, indicate that this function was not entirely lost, while the moderate evidence of old hæmolysis in the hæmal glands would further bear this out. But these glands show an excessive number of phagocytes full of intact red blood cells which is readily explained by the results of Warthin's work. He concludes from his experiments with sheep that hæmolysis and leukocyte formation are the functions taken up by the hæmal glands after splenectomy. But the hæmolytic action of these glands exceeds that of the normal spleen, and the resulting anæmia is compensated for by increased activity on the part of the bone-marrow. The marked fall in the blood count after splenectomy was due then to this excess of hæmolymphatic hæmolysis over that of splenic.

No iron pigment could be demonstrated in the liver and kidneys. This fact, together with the paucity of megaloblasts in the bone-marrow, indicates that the anæmia was not primary in type. While the hyperactivity of the hæmolymph glands would explain the anæmia which undoubtedly existed before the accidents, the intestinal hemorrhages following them reduced the patient to his state on entering the hospital.

Pathological Diagnosis. Dislocation of the spleen with marked elongation and torsion of its vessels, and thrombosis. Chronic passive congestion and fibrosis of the spleen. Interlobular atrophic cirrhosis of the liver. Chronic passive congestion with beginning central cirrhosis of the liver. Chronic passive congestion of rest of portal system. Compensatory hyperplasia of hæmolymph nodes. Hyperplasia of the bone-marrow. Secondary anæmia. Moderate diffuse emphysema of the lungs. Tuberculosis of bronchial and tracheal glands.

My thanks are due to Dr. W. T. Howard, Jr., for the use of material and for his interest in the work.

3. General Summary. The patient's previous history of pallor, digestive disturbances, and pain in the splenic region, symptoms of which there is a history for a number of years, strongly suggest the

early stage of a splenic anæmia. The frequent urination and the pain in the splenic region also would imply a long-standing dislocation of the spleen. This is corroborated by the enormous elongation of the splenic vessels. Whether or not all the twists in the splenic veins occurred at the time of the two accidents, it is impossible to state, but it is at least more reasonable to suppose that from their multiple character they did not all occur at one time, but that some had existed previously.

The thrombosis of the splenic vessels must have been the cause of the sudden enlargement of the spleen while the patient was in the hospital. The lack of organization in the thrombi of the vessels removed at the splenectomy proves this.

If Banti's stage of splenic anæmia is limited only to those cases in which the cirrhosis is a terminal manifestation we cannot include our case in that category, for the cirrhosis of the liver is certainly of as long standing as the fibrosis in the spleen. We prefer to regard the case as one of splenic anæmia associated with a cirrhosis of the liver, not as a result of the process in the spleen, but rather accompanying it.

The enormous elongation and torsion of the vessels associated with the dislocation of the spleen possess a very probable etiological significance in this case.

SARCOMA OF THE SMALL INTESTINE:

WITH EXTENSIVE METASTASIS IN THE HEART MUSCLE AND EMBOLISM OF THE SUPERIOR MESENTERIC ARTERY.[1]

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In 1900 I published five cases of sarcoma of the small intestine and collected about fifty other cases from the literature.[2] Since that time we have had in the hospital ten further cases of this disease, in addition to which a small number of cases has also been reported by various other observers, most of which are collected in the valuable monograph of Lecêne[3] published during the past year.

In none of the cases which I have seen or which I collected from the literature was there any note of metastatic deposits in the heart,

[1] Demonstration at the meeting of the New York Pathological Society, December 14, 1904.
[2] The American Journal of the Medical Sciences, 1900, vol. cxx. p. 309.
[3] Les tumeurs malignes primitive de l'intestin grêle, Paris, 1904; Mittheilungen aus den Grenzgebeiten, Bd. vii. Heft 415.

and this is the first time we have encountered such marked involvement of the heart in any case of growth.

The patient, a male, aged thirty-five years, was admitted to Mt. Sinai Hospital on July 26, 1904, and died on August 19th. He was under the care of Dr. Gerster, to whom I am indebted for the use of the clinical history.

Family History. Presents no points of any importance.

Previous History. Four years ago he had an attack of pain in the hypogastrium lasting two days, which was relieved by morphine. He had no chill at this time and no pain in the loins, nor along the course of the ureter. The pains did not radiate; the urine was clear. Three and one-half years ago he had a second similar attack; two years ago he had a third attack lasting five weeks, for the last two weeks of which time he was taken care of in another hospital.

Present History. Since the last attack he was well until four months ago, when the hypogastric pain returned, and he developed pains in the left loin, sticking in character. These pains did not radiate downward. For the last two months he has had pain at the end of the penis during urination; the stream is never interrupted and the urine is always clear. He had been getting up once every night for the past three weeks to urinate. Three weeks ago he began to vomit all his food; this lasted one week. He has not been jaundiced. Has been in bed since the vomiting began. He thinks he has lost fifteen pounds in the last year.

Examination on Admission. The external lymph nodes are not enlarged, except those in the submaxillary region, which are slightly enlarged.

Heart. The dulness extends from the right border of the sternum to the nipple line; the apex-beat is not made out; the sounds are weak; the first apical sound is short, with very little muscular quality. *Pulses* are equal, regular, rapid, of poor force, markedly dicrotic. There is marked sclerosis.

The lungs present the signs of emphysema, with slight bronchitis.

The liver is enlarged to one and one-half fingers below the free border of the ribs.

Spleen not palpable.

Abdomen is held rigid, is generally tympanitic; there is marked tenderness just above Poupart's ligament on the left side.

Genitals show no changes.

There is no œdema of the legs.

Rectal Examination. Prostate negative. Commencing about one and one-half inches above the prostate and extending up beyond the reach of the finger is a mass feeling about as large as an orange, extending somewhat more to the left than to the right, movable, nodular. Pressure exerted upon this causes pain to be felt at the end of the penis. The tumor bulges somewhat into the anterior wall of the rectum.

The patient was operated upon on July 30th by Dr. Gerster. On opening the abdomen, the appendix was found adherent to a large mass filling the true pelvis. The mass itself was found to be a tumor involving the intestines, which would not permit of removal.

From the subsequent history of the case we shall give a few data only:

August 2d. Vomiting frequent; temperature 100° to 101°; no pain.

5th. Bowels moved; no vomiting since the fifth day after operation. Temperature 101.6°.

6th. Flatness over the right lower lobe; breath sounds distant and blowing; crepitant rales.

8th. A fecal fistula has developed.

On the 13th large lymph nodes were felt in the left groin. There developed at the base of the left lung dulness and a few mucous rales. The patient died on August 19th.

The urine was usually of specific gravity of 1020 to 1030, contained a trace of albumin, a few red blood cells, pus cells, mucus, and many bacteria.

The autopsy examination was restricted to an examination through the wound.

Abstract of the Autopsy Report. Thymus. No traces found.

Œsophagus. Moderately dilated.

Lungs. In the left pleura there is a large amount of fluid; in both lungs there are hemorrhagic infarcts and areas of new-growth. The bronchial nodes are enlarged, pigmented, and infiltrated. The posterior mediastinal nodes are also infiltrated.

Heart. Weight, 510 g. On the surface of the right ventricle and auricle are several rounded hemispherical prominences, white in color, 2 to 3 cm. in diameter. All the chambers are dilated. The foramen ovale is slightly open. The wall of the right ventricle is thickened. The color is grayish-red, particularly near the septum. There are a few small parietal thrombi. The tricuspid valve is thickened. Left ventricle: The cavity is dilated; the wall is 2 cm. thick, is gray in color, with a slight admixture of red; it is rather friable. There is evidently a very marked diffuse infiltration by new-growth. The mitral valve is thickened; behind one of the papillary muscles is an adherent decolorized thrombus.

The *aorta* and *coronary arteries* show fatty changes.

Spleen. Enlarged; marked perisplenitis; a few anæmic infarcts; congestion.

Liver. Weight, 1⅞ kilos. On section congested, cloudy, fatty; a few small areas of diffuse growth in the right lobe near the free border. Gall-bladder: Wall, ½ cm. thick; diffusely infiltrated by new-growth.

Kidneys. Several anæmic infarcts in each kidney; also areas of new-growths, which shade gradually into the kidney tissue proper; a few of these are elevated; pelvis dilated; ureters negative.

Pancreas and *adrenals* negative.

Bladder. There is infiltration by new-growth of the posterior wall up to the mucosa; there are several blackish areas on the posterior wall; the vessels of the trigone are injected.

Stomach dilated; wall acutely inflamed.

Intestine. In the duodenum there is infiltration of the wall, white in color, for a length of 10 cm. The intestine is dilated at this point. Below this infiltration, between it and the main tumor, there are several places at which the intestine is infiltrated for short distances and is dilated. At a distance of 120 cm. from the beginning of the jejunum there is a very large dilatation of the intestine due to infiltration of its wall by new-growth. This dilatation fills the entire true pelvis. The growth is white in color, soft. The greatest thickness of the wall of the dilated intestine is 1½ cm. On section the growth is white and soft. The inner surface is necrotic and ulcerated. It is adherent to the bladder, which is infiltrated by contiguity, and is also adherent to the sigmoid flexure and coils of small intestine. It lies above the prostate, not attached to it.

The *mesenteric nodes* are enormously enlarged; on section they are white in color, with yellow areas. The cæcum is dilated. The rest of the colon is collapsed.

Superior Mesentery Artery. Just above the bifurcation there is an adherent, partially decolorized thrombus which extends slightly into both divisions of the vessels, not entirely filling their lumina.

Microscopic Examination. A large amount of material from the case was studied, but only a brief report will be given here. The primary tumor of the intestine proved to be a lymphosarcoma. The lungs, heart, kidneys, spleen, mesenteric nodes, liver, gall-bladder, and urinary bladder wall showed metastases. The pancreas and adrenals showed no metastases. In the wall of the left ventricle and in the septum there was found to be a very extensive infiltration by new-growth, the individual muscle fibres being crowded apart by the cells of the new-growth (see illustration). There was marked degeneration of muscle fibres. The spleen and liver showed marked pigmentation. In the lung there were chronic interstitial changes and small purulent foci. The kidneys and liver showed parenchymatous degeneration. In the intestine the growth was found to begin in the submucosa and to infiltrate between muscle bundles.

There are several points of interest about this case:

1. It is a very good example of the dilatation which often occurs with lymphosarcoma of the intestine. In this case the dilatation was more marked than any other I have ever seen.

2. As is quite common in these cases, there was no ascites, and no enlargement of the external lymph nodes.

3. The growth in the pelvis with adhesion to the bladder is a characteristic of some of these tumors, which deserves more attention from a clinical standpoint than has heretofore been given it.

4. The duration of the disease is the longest that we have noted in any case. Although from the standpoint of the findings in the case we would ordinarily have diagnosed a lymphosarcoma of the small intestine, the long duration of the case led us to suspect a non-malignant neoplasm or tuberculosis of the intestine with adhesions to the bladder. Baltzer put the duration of the disease at from two weeks to one and three-fourths years, and said that most cases died in nine months. Rutherford described one case with a duration of two and one-half years. In the series of cases which we had collected, this last-mentioned case was the longest one.

5. The embolism of the superior mesenteric artery caused no appreciable phenomena clinically, nor did it cause any change in the intestine. This was probably due to the fact that it was quite recent and that the embolus did not completely close off the lumen of the vessel.

6. The involvement of the heart was the most striking feature of the case. Although a note was made on admission to the hospital that the first heart sound had very little muscular quality, there was no suspicion that the heart was involved by new-growth.

The illustration, for which I am indebted to Dr. Mandlebaum, shows the infiltration in the heart at a magnification of 250 diameters.

THE CLINICAL ASPECTS AND DIFFERENTIAL DIAGNOSIS OF APPENDICITIS AND TYPHOID FEVER.

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of Boston, Mass.

In this short paper it is not our intention to give a detailed description of all the clinical manifestations belonging to typhoid fever, or appendicitis, or to the various forms that these two abdominal affections may take on. Our aim is to consider the principal elements of a differential diagnosis rather than to give a long description of the symptomatology; in other words, we have more in view the study of abnormal forms of typhoid fever which may closely simulate appendicitis and thus cause much hesitation on the part of the physician and surgeon. A typhoid fever following its normal and regular evolution without complications only distantly resembles a typical attack of appendicitis, so that it would be difficult to mistake one for the other; but there exist, on the contrary, certain abnormal forms, and they are by no means infrequent, in which considerable difficulty in diagnosis arises. In the first place the mild forms of typhoid should be mentioned. It is well known that in some of these the temperature is not very high, the rose spots may be so discrete that they are usually unnoticed, while the bronchitis may be wanting and the headache very slight. In these cases of mild intensity the diagnosis of typhoid will be based exclusively on a slight pain that may be made manifest by palpation of the right iliac fossa, accompanied by a slight headache and a trifling enlargement of the spleen. Diarrhœa frequently is absent and may even be replaced by constipation. Then again it should be recalled that some of these cases are so slight that the patient does not feel sick enough to remain in bed; in other words, walking typhoid.

The same may be said of cases of attenuated typhoid, which are apyretic and can only be diagnosed by the Widal reaction. All these cases may simulate the commencement of appendicitis where the pain in the right iliac fossa is very slight, while other symptoms indicating a disturbance of the gastrointestinal tract are present to a certain degree, and all surgeons are aware that this form is not exceptional. There are cases of appendicitis commencing by symptoms of enterocolitis, accompanied by a persistent diarrhœa, and, on the other hand, there are instances of appendicitis with a very slow and almost apyretic evolution, commencing insidiously, and where large and extensive plastic exudation, with considerable abdominal distention, easily conceals the purulent focus. Consequently, under these circumstances, an erroneous diagnosis of mild

typhoid is quite possible. And since typhoid fever, even very mild and apyretic, may suddenly become complicated by a superacute perforating peritonitis, just as is met with in cases of insidious appendicitis, it can be readily seen how a diagnosis may be difficult. On the other hand, certain serious types of typhoid may lead to a confusion in the diagnosis just as in the case of the lighter types. I do not intend to refer to the serious forms accompanied by marked nervous or hemorrhagic phenomena, in which the diagnosis falters between meningitis, acute miliary tuberculosis, or any other acute affection other than appendicitis. But there exist certain serious forms, inasmuch as there is an early and intense reaction of the intestine on the peritoneum with a predominance of serious abdominal symptoms, more than by the symptoms furnished by the general condition of the patient. Now it may be stated that for these forms, as well as for others, nothing is more variable than the intensity of the various symptoms of typhoid; abdominal distention, pain in the right iliac fossa, and diarrhœa may reach the maximum point, while the rose spots may be extremely discrete, hardly any bronchitis, and nervous phenomena practically *nil*. Many authorities appear disposed at the present time to admit that a marked eruption of rose spots is more apt to belong to a typhoid of medium or slight degree; and, on the other hand, it is well known that the rose spots never appear before the seventh or eighth day of the disease. Consequently, when in presence of one of these cases having a predominance of marked abdominal symptoms, a hesitancy in diagnosis between typhoid and appendicitis will be quite possible, at least during the first week of the malady. And *a fortiori* one may be extremely embarrassed in cases presenting a true peritonitis, or one resulting from a perforation. It may be added that it is not rare to meet typhoid with a fever of 39° or 40° C. during the first twenty-four hours, and these cases with a sudden commencement render the diagnosis a difficult matter.

To sum up, there exist many cases in which typhoid and appendicitis take on similar characters and are extremely difficult to distinguish one from the other, and these usually represent the forms of medium intensity of both these diseases; there are a certain number of symptoms which are common to both, especially pain in the right iliac fossa with or without gurgling on pressure, abdominal distention, a rise in temperature on the very first day of the disease, enterocolitis, and diarrhœa. With these symptoms others may be met with which belong distinctively to each of these affections. The absence of pulmonary symptoms, rigidity of the abdominal muscles, the condition of the pulse, and symptoms of peritonitis would be in favor of a diagnosis of appendicitis when they exist. Bronchitis, severe headache, insomnia, epistaxis, rose spots after the eighth day, and hypertrophy of the spleen point to typhoid fever. We here merely mention the symptoms common to

both diseases, or belonging to them in particular, and will study their semeiological value later on.

From this cursory description it becomes evident that the differential diagnosis between typhoid fever and appendicitis is frequently a very difficult matter. A positive diagnosis of either of them may be difficult in those frequent cases in which several of the cardinal symptoms are wanting. As to the differential diagnosis it is sufficient to review the numerous errors which have been made to convince one's self of the extreme difficulty in which the clinician is frequently placed. Thus, for example, only to point out one of the principal causes of mistake an appendicular colic may be taken for one due to nephrolithiasis, gallstones, intestinal occlusion, or lead colic. Appendicitis has also been mistaken for an acute tuberculous peritonitis, more frequently for a salpingitis or typhoid fever. These cases we will not consider because we only intend to study the differential diagnosis of appendicitis in general, and we will limit ourselves to the differential diagnosis with typhoid fever. In a case reported by Bayet, the patient was a young woman who was suddenly taken with febrile phenomena, accompanied by a slight indefinitely localized pain in the right iliac fossa. On account of the large size of the right tube, a diagnosis of acute catarrhal salpingitis was made, and this diagnosis appeared all the more justified because, under the influence of quiet and appropriate treatment of this slight local lesion, the temperature fell in two or three days. The patient appeared to be cured when she was suddenly seized by an acute pain at McBurney's point with serious general symptoms and a high temperature. As the diagnosis of appendicitis did not appear to be a matter of doubt, the patient was operated on the next day. The appendix removed was of normal size, slightly hyperæmic, and contained a clot of blood, an important fact to which we will return later on. In spite of the operation the disease became worse and the patient died a few days afterward. Autopsy showed characteristic ulcerations of typhoid fever. The night before death Widal's test was positive.

Several other similar cases have been published, and a number of authorities have only mentioned the possibility of a mistake in differential diagnosis, without going into any details.

We must not lose sight, in the appreciation of the elements of the differential diagnosis, that appendicitis is a disease that must be recognized at its very beginning. Upon the precociousness of this diagnosis depends the indications for operation and the patient's life; consequently, when one is in the presence of one of these difficult cases that we have already considered, and when one hesitates between an abnormal form of typhoid fever and appendicitis, too much precaution cannot be taken in making the diagnosis, and this diagnosis should be based on the greatest number of

elements possible. These elements of differential diagnosis may be classified under four heads, as follows:

(1) An analysis of the clinical symptoms; (2) examination of the urine; (3) examination of the blood; (4) Widal's serodiagnosis.

The clinical symptoms leading to a differential diagnosis are essentially variable, and no one, taken alone, is pathognomonic. Consequently, one should take into consideration the ensemble of these symptoms rather than any one of them; and in spite of such grouping, the clinical aspect of the disease is, under certain circumstances, absolutely insufficient, and, in order to come to any conclusion, one should take into consideration the ensemble of the other symptoms, to which we will refer later on.

It is, however, often possible to obtain considerable information from an attentive study of the case, which will allow of making a serious presumption, if not a certitude of the diagnosis. Among the symptoms common to the two diseases, pain in the right iliac fossa is the most constant, but its value is not an absolute one and is not sufficient to enlighten the diagnosis. It is true that this pain, in cases of appendicitis, is more apt to be sudden and to merit the name of appendicular colic, while the pain of typhoid is more inclined to be insidious in its evolution, shooting and not pungent, as in appendicitis. Taking into consideration that in the majority of cases spontaneous pain is less frequent and less acute in typhoid, that palpation of the abdomen gives rise to sudden and exquisite pain in appendicitis, and that the localization of this pain is different in each affection, one may draw some conclusive evidence in favor of one or the other. In typhoid it is spread out through the entire iliac fossa, while in appendicitis it is quite limited, and in many cases is particularly intense over McBurney's point. Now, although this is true in a large number of cases, the characters of the iliac pain present numerous variations, and the differences that we have pointed out are subject to many exceptions, which may lead a physician into error if he is not on his guard.

In the first place the pain in the right iliac fossa may be absolutely wanting, or very attenuated and temporary, both in certain forms of typhoid fever as well as in some cases of appendicitis. It is well known that the symptomatic pain is very variable in appendicitis, not only in its acuity, but also in its localization, and a certain number of cases have been reported where its greatest intensity was found in the left iliac region. In quite a few cases the greatest point of pain will be found either above or below McBurney's point. Then again pain in the right iliac region may be induced by other entirely different affections, such as a calculus or a salpingitis.

With the pain we should also mention intestinal gurgling on palpation of the iliac fossa, but only for the purpose of eliminating it as a diagnostic sign. It has absolutely no value, and simply indicates the presence of gas in the intestine.

Muscular rigidity, on the contrary, was for a long time considered as a most important symptom, always speaking in favor of appendicitis, particularly when the process had gone on to suppuration. At the present time it is impossible to retain the importance that was formerly given to this symptom, because if one palpates an iliac fossa, containing some lesion, pain nearly always will be produced, immediately followed by a reflex contraction of the abdominal wall, which instinctively puts itself on the defence. Now, one can readily imagine that the pathogenesis of the symptom of muscular rigidity is a very general phenomenon. When the distention is not very pronounced in a case of typhoid, palpation may produce a contraction of the abdominal walls, and, on the other hand, in some cases of clinically characteristic appendicitis, verified pathologically, this symptom may be very slightly marked, or even be absolutely wanting. Consequently, it may be said that the absence of muscular rigidity, which is ordinarily the case in typhoid, may also exist in appendicitis, and the semeiological value of this symptom is only relative.

Cutaneous hyperæsthesia has not much value.

We do not believe that there is any more certitude in the temperature curve, the height of the fever in the commencement, and the symptoms of enterocolitis, because in subacute or chronic appendicitis the temperature chart looks very much like one of typhoid, and even if there existed very distinct differences in this respect, this symptom would only have a relative value in the question we are now considering, because it only begins with the commencement of the disease. I have already said that there are cases of appendicitis without fever just as there are cases of apyretic typhoid.

The best guide, when they are present at the commencement, are the symptoms of enterocolitis. In many cases appendicitis is accompanied by constipation at the onset, but to this rule there are numerous exceptions, and diarrhœa may be quite as persistent and as abundant as in typhoid. Inversely it is not infrequent to meet with cases of typhoid in which diarrhœa is absent and is replaced by constipation during the greater part, if not the entire duration, of the disease. As to vomiting it is observed at the onset of typhoid as well as in appendicitis, and gives no clue to the diagnosis.

From all this it goes to show that the clinical symptoms common to both diseases may be present in either one or the other, presenting very similar characters which will prevent one from utilizing them in making a differential diagnosis. When the typhoid presents no other symptoms, it will be clinically impossible to diagnose it, but it is only right to say that in practice such cases are not often met with. In typhoid from the very beginning the headache is extremely severe, and an important character is that it becomes better as time goes on and the abdominal symptoms increase in intensity. In appendicitis headache is practically absent, and when it does exist

it does not follow this same progressive diminution. Bronchitis is also wanting in appendicitis, but it also is not at all marked during the first few days of typhoid. The epistaxis, when it occurs at the beginning of typhoid, is more valuable, but insomnia during the first few days appears to have, in our way of thinking, a still greater importance; associated with an intense headache it certainly pleads greatly in favor of the latter affection. The appearance of a marked eruption of rose spots is a certain sign of typhoid, but, unfortunately, this does not occur before the seventh or eighth day of the disease, and I have already pointed out the necessity attached to an early differential diagnosis. It should be remembered that in a number of cases the rose spots may be absent, either really so, or they are so discrete and ephemeral that they pass by unnoticed. Hypertrophy of the spleen is possessed of no pathognomonic value and simply indicates the degree of the infectious condition of the patient, and it is well known that certain cases of appendicitis take on the characters of hypertoxic infectious diseases. A rapid pulse, prostration associated or not with a variable degree of delirium, like a pinched expression of the face, may be observed in many cases of serious infection, as in typhoid.

It would appear to result from the above considerations that no single symptom taken in itself is possessed of a pathognomonic value, but it is not at all doubtful that when taken altogether these symptoms give a positive diagnostic indication, infinitely more precise than the analysis of any one of these symptoms taken singly. And there exist a certain number of cases in which, by reasoning, a diagnosis may be made with only a small chance of making a mistake. But, on the other hand, there are other patients in which the affection assumes such an aspect that it is absolutely impossible to determine its nature with certitude, and these are those instances where the symptoms appear to multiply themselves to such an extent that the physician is absolutely lost. The localization of the pain, exactly on MacBurney's point, the sudden and dramatic commencement, accompanied by high fever, vomiting, hiccough, in a word all the symptoms of a very acute appendicitis, have been observed with the greatest distinctness to such a point that an operation has been decided upon and immediately done, when in reality the case was one of typhoid, the patient possessing a perfectly normal appendix. Cases of this description defy all analysis, but fortunately there are others in which the study of the symptoms in their ensemble makes a less difficult interpretation. In point of fact, in the majority of cases the abdominal symptoms are those which are foremost in the clinical picture of typhoid, while, on the contrary, the gravity of the general condition often overshadows from the very commencement all other signs found upon local examination, when the case is one of appendicitis. Without making this an absolute rule and without forgetting those cases of typhoid,

happily infrequent, in which from the very start the general symptoms become alarming, it is nevertheless quite permissible to suppose that we have here a serious element for appreciation in the diagnosis; given an equal gravity, an appendicitis manifests itself by more accentuated general symptoms than a typhoid, especially during the first week, and the abdominal symptoms are, on the contrary, less marked in the latter than in the former.

The commencement of the symptoms, their manner of acting in the beginning, should also to a certain extent be taken into serious consideration. Generally speaking, the commencement of a typhoid fever is insidious and the temperature curve is progressively ascending; while usually in acute appendicitis, on the contrary, the commencement is sudden and the fever high from the start. However, we know that this rule has many exceptions. The evolution of the symptoms may also, under certain circumstances, help in the diagnosis. Typhoid has usually a regular and cyclical evolution, while that of appendicitis is capricious, often proceeding by successive steps and subject to recurrence. However this may be, it is clearly evident that the clinical symptoms taken singly, or considered in their ensemble, have occasionally only a very relative semeiological value, and their analysis in certain cases only gives a more or less serious presumption in favor of one or the other of these two diseases. In some exceptional cases the symptoms, although very rationally interpreted, will be in absolute disaccord with the true diagnosis.

The examination of the urine, which is absolutely necessary in all cases, even the simplest, has a still greater importance when the case is a doubtful one. In all acute febrile diseases albuminuria is usually present and consequently may be met with both in typhoid and in appendicitis. However, if one will observe that at the time the first symptoms appear appendicitis is a local affection, while, on the other hand, the first positive symptoms of a typhoid indicate a diffusion already realized of the typhoid toxins throughout the entire economy, it would seem probable that, in the beginning, at least, albuminuria will be more frequent and more pronounced in typhoid than in appendicitis. In the former the kidney is already under the influence of the toxin, while in the latter the toxin has not as yet had time to be produced. Now this remark, which may at first sight appear somewhat theoretical, is fully confirmed clinically. It is with intent that we insist upon the fact that we are referring to the commencement and later, when the appendicular lesions have allowed the toxins to become diffused throughout the economy on account of the close pocket in which they are manufactured, susceptible of completely destroying organs such as the liver, and which can result in death with all the symptoms of a hyperacute poisoning, large quantities of albumin will be found in the urine.

Indicanuria is of an absolutely secondary diagnostic value, because indican may be found in the urine in all infectious conditions of the digestive tract and an ordinary attack of indigestion is quite sufficient to make it appear. According to a certain number of authors indicanuria is never absent in typhoid, but others have upheld that it is absent in the commencement and its maximum reached during convalescence. On the other hand, a large quantity of indican may be found in the urine during the first few days of appendicular infection, and, consequently, this is a differential symptom which may, to a certain extent, be utilized; but I would repeat that, according to my way of thinking, its importance is only secondary. Certain indications may also be derived from the toxicity of the urine, and this has been especially studied in children during appendicitis by Lannelongue and Gaillard. From their researches it becomes evident that in appendicitis the urinary toxicity increases at once, at the same time that the density increases and its color becomes dark. On the contrary, in typhoid, the toxicity becomes above the normal, principally at the beginning of convalescence at the time when the patient is undergoing a urotoxic crisis, which becomes manifest by a considerable increase in the quantity of urine, corresponding to a discharge of extractive substances from the organism.

Blood examination may give in many cases valuable hints and the importance of hæmatology in the study of these diseases is greatly appreciated at the present time. I do not believe that it is too much to say that the recent method devised by Ehrlich, in allowing one to classify the various forms of leukocytes, not only according to their forms in general and their nuclei, but according to their affinity for basic or neutral coloring acids, has given a new impulse to the histology of the blood and has completed the study of the characters that this tissue presents in infections and intoxications. But it must be admitted that this study, which for the various leukæmias is still incomplete, has just been begun in the greater number of other affections. It is far from our intention to make a detailed study of the condition of the blood in appendicitis and typhoid, and we will only refer to certain characters which may aid in making a differential diagnosis. In appendicitis the clot is found microscopically to be composed of an abundant fibrinous network, and this aspect, which is found in all inflammatory diseases, does not exist in septic affections like typhoid; this characteristic may consequently be utilized in a certain number of cases. However, on the very account of its generality, one can readily see that it can only be utilized in certain cases where the differential diagnosis is faltering between the two diseases which we are considering. It consequently will be absent in certain forms of appendicitis which very rapidly take on the aspect of septicæmia, or in some instances of typhoid complicated by inflammatory accidents

and commencing with them, as, for example, pneumotyphoid or pleurotyphoid. The examination of the serum does not give any marked result, but sometimes in typhoid the clot is small and the serum proportionately abundant; occasionally also in certain hemorrhagic forms of this disease the serum takes on a hæmoglobinuric color, but these are characters far too inconstant to take seriously into consideration.

The study and the numbers of the figured elements have a much greater importance, and it is known that in inflammatory infections, like appendicitis, especially when it is of the suppurating variety, the number of leukocytes increases very greatly. On the contrary, in typhoid fever and septic diseases, without inflammatory complications, the count remains normal; and there may even be a hypoleukocytosis. This sign accompanied by an absence of the fibrinous exudate takes on a very great diagnostic value which has been pointed out by Hayem: "In cases of typhoid without any inflammatory complication he points out it is a characteristic of the disease; the absence of a fibrinous reticulum and the small number of leukocytes visible in the field of the microscope." (Lenoble.) Muehleck has also found a diminution in the number of red cells, as well as their value in hæmoglobin in many cases of typhoid fever. To sum up, it may be said that an examination of the blood should never be neglected in doubtful cases; unless there are inflammatory complications in typhoid, or a hyperacute septicæmia of appendicular origin, it will often give very useful indications for the differential diagnosis of these two affections.

Of all the means of investigation at our disposal the serodiagnosis of Widal is assuredly the one that gives the most certain results other than the clinical symptoms. In all doubtful cases of typhoid fever the research for this sign has a capital importance and in itself alone it will allow one to distinguish a mild typhoid from slight gastric disturbance, a serious typhoid from a meningitis, or all other affections which may simulate it. In many cases where there is any hesitancy in the diagnosis between a typhoid and an abnormal appendicitis the serodiagnosis will settle the question, and where the result is positive this sign has absolute certainty. However, without wishing to decrease the great value of this reaction, it is necessary to bear in mind that it is occasionally absent under certain circumstances, and it is precisely these that must be looked for, particularly in those cases where the diagnosis of appendicitis is faltering.

In the first place there are some few subjects whose serum does not agglutinate, or only very slightly so, the culture of Eberth's bacillus, although they are manifestly the possessors of typhoid. Such cases are reported from time to time, but they are generally speaking exceptional. As a rule, the seroreaction does not become manifest before the seventh or eighth day of the disease, and it

cannot be too often repeated that when there is any confusion with appendicitis it is at the commencement of the affection that the problem must be solved; to wait a week before deciding to operate would cost many lives. It consequently becomes evident that, in the particular case which we are considering, the importance of Widal's test is much less and this sign will practically lose all its value when it appears late in the affection.

It is not at all infrequent to meet with tardy agglutination, only appearing a fortnight or eighteen days after the commencement of the typhoid, and in these cases the time at which the serum test becomes positive naturally takes away the greater part of its importance. On the other hand, it would seem from the most recent works on the subject that agglutination is all the more apparent the more the typhoid retains a medium intensity and a normal evolution. In serious typhoids the agglutinating reaction is often *nil* or hardly marked, which is another circumstance calculated to cause a diagnostic error. In point of fact it is known that the patient's serum, under the influence of typhoid infection, having acquired the property of agglutinating typhoid cultures, will retain this property for a considerable length of time after recovery from the disease, and this agglutinating property may show itself a year, or even more, after typhoid. Now, as an appendicitis may develop consecutively upon a typhoid after a more or less long lapse of time, it is easily conceived that before rejecting the diagnosis of appendicitis in cases where the serum test is positive, one should always obtain a history of the morbid antecedents of the patient, and with this reserve Widal's test possesses a diagnostic value of the highest order and one that should never be neglected in doubtful cases.

In the preceding pages we endeavored to ascertain upon what elements a differential diagnosis between typhoid and appendicitis might be made, and we will now endeavor to apply what we have learned to certain cases which may be met with clinically. We will limit ourselves to the principal ones, voluntarily leaving aside the very numerous intermediary types whose description would extend the subject matter of this paper without profit.

One may find himself in the presence of a slight infection, characterized by a gastroenteritis of medium intensity with headache, moderate fever, and pain in the right iliac fossa. Under these circumstances the development of the disease can only rarely help the clinician to decide upon the diagnosis which may very well be faltering between a mild typhoid fever in the beginning, a simple gastrointestinal intoxication, or an attenuated appendicular colic. Muscular rigidity and cutaneous hyperæsthesia are usually wanting and the localization of the pain will be difficult. Given such a symptom-complex, the degree and progress of the headache, insomnia, if the disease is at its commencement, the appearance of

rose spots and serodiagnosis, if the disease has attained the end of the first week, will be, according to our way of thinking, the principal signs upon which one may base the diagnosis.

Under other circumstances, a physician may be called to a patient presenting all evidences of a serious disease and in which the predominating symptoms will be abdominal. The case will not look like one of true peritonitis, but the abdomen will be distended with acute pain in the right iliac fossa and either a profuse diarrhœa or an obstinate constipation will be present. Under these circumstances the development of the symptoms is valuable and will be completed by the elements furnished by clinical examination, as well as by the examination of the blood and urine, as we have already pointed out, and if Widal's sign is positive the diagnosis becomes evident. In other cases one will find a confirmed peritonitis, in which case the pathogenesis and etiology must be ascertained. Formerly two distinct types of acute peritonitis, occurring suddenly during a febrile malady were distinguished; one group was called peritonitis by propagation, the other from perforation, and to each of these modalities was connected a special clinical invasion. At the present time it is known that a large number of the so-called peritonitis cases from propagation are due to perforation, although the latter may be extremely minute, but existing nevertheless, and it may be said that the form by propagation is very infrequent, if in reality such cases really exist.

Let us suppose that the case presents the ordinary symptoms of a perforating peritonitis, the question will at once arise as to whether it is due to a ruptured appendix or a perforated typhoid ulcer. This is occasionally a very delicate matter to determine, all the more so because in other diseases, in gastric ulcer, for example, the same condition may arise. But the difficulty is quite great enough for us to limit ourselves to the consideration of the two diseases which we are studying. In some cases the differential diagnosis will be quite possible if the age of the disease is considered, because a perforation of a typhoid ulcer infrequently takes place before the second week and rarely occurs after the third. Generally speaking, perforation of the appendix is much more precocious and may even take place during the first few hours after the development of the symptoms, and the disease may at once show itself with all clinical characters of perforation. In doubtful cases the initial localization of the pain should be carefully ascertained, as well as the condition of the spleen and lungs; one should watch for the possible appearance of rose spots, as well as the existence of a leukocytosis and the presence or absence of Widal's sign in those cases where the disease has lasted for a week or more. The time at which the perforation occurs, and the preceding evolution of the disease, as well as the serodiagnosis, will, we believe, serve as the principal diagnostic elements in these cases

which rapidly take on the role of toxæmias more than inflammatory processes.

A typhoid complicated by perforation rarely gives rise to a circumscribed peritonitis, and usually one will be dealing with a diffuse inflammation of the peritoneum, while a localized peritonitis is more frequently observed in perforation of the appendix. Cases of periappendicular abscess are rarely mistaken for typhoid, because they are easily diagnosed by the muscular rigidity which, under these circumstances, is rarely wanting, as well as by the temperature chart, the small, rapid pulse, and other signs of intra-abdominal suppuration. If there is any doubt, examination of the blood and urine will have no very great importance, because, in these inflammatory processes, with a tendency to suppuration, leukocytosis will almost always be present, whether the case is one of suppurating appendicitis or a typhoid complicated by some inflammatory lesion. Consequently, it is well to base one's self more upon the elements furnished by the clinical evolution of the disease and to the Widal test.

So far we have compared appendicitis and typhoid fever, but we should now consider those cases where both diseases simultaneously exist in the same patient; in other words, can an appendicitis complicate typhoid fever? The reality of this pathological association is not doubtful, although the frequency is a much discussed point. A certain number of authorities are of the opinion that the appendix is almost always involved in typhoid, but that the lesions found are those of slight inflammatory nature not sufficiently marked to give rise to symptoms of a true appendicitis. On the other hand, there are others who classify the cases under two separate heads, namely, those in which the congestion of the appendix remains moderate, and, secondly, those in which a true appendicitis arises during the progress of the typhoid. It is quite permissible to admit that these instances, at the present time well established, cannot be extremely infrequent, and they are, for that matter, possessed of a very rational interpretation. In the first place the lymphatics of the cæcum and appendix are in free communication with those of the end of the ileum, and thus a direct extension of the lesions may take place. But besides this the histological structure of the appendix is in every way comparable to that of the small intestine, and in particular a large amount of lymphoid tissue is to be found which, at certain parts of the organ, may form miniature Peyer's patches. It would, consequently, seem quite natural that this lymphoid tissue can be the seat of an intense hyperæmia, and even go as far as an ulcerative process when Peyer's patches present marked lesions. Now, it is well known that epidemics of appendicitis very frequently coincide with epidemics of general infectious diseases, and many authorities have pointed out the great frequency of appendicitis during epidemics of la grippe, while others have given a general infectious origin to appendicitis.

Among the general diseases it is quite natural to suppose that typhoid should enter for a large share, and in point of fact it can be shown that certain typhoids become complicated, principally during the acute period, by accidents having a distinct appendicular origin. In other cases the appendicitis arises during the decrease of the disease, or during convalescence, and these are the instances that Dieulafoy has described under the appropriate term of para-typhoid appendicitis. The patient hardly recovers from the acute symptoms of the typhoid, he has hardly entered into convalescence, when suddenly characteristic signs of acute appendicitis appear. Lastly, it is not infrequent to meet with appendicitis in patients who have had typhoid one or more years previously.

It now remains for us to examine the mechanism of appendicular accidents occurring in typhoid. Generally speaking this mechanism is little known. In mild cases, where the lesion of the appendix is indistinct, most authorities believe that the symptoms observed are referable to a mere congestion of the organ. This inflammation arises from direct propagation, or by lymphatic propagation, but the process does not go as far as ulceration, and clinically speaking there is not appendicitis. In two cases of typhoid fever during convalescence I have removed the appendix, believing from the symptoms that the organ was the seat of an inflammatory process, and, although macroscopically the organ appeared normal, yet, upon section, its cavity contained a clot, due without any doubt to a hemorrhage arising in Peyer's patches, which microscopically showed evidences of ulceration and which, in all probability, would have resulted in perforation of the organ had the process continued. It may be that the clot, playing the part of a foreign body, gave rise to the symptoms of appendicitis in these patients, but I feel, nevertheless, that at least in these two instances the appendix was better in a bottle than in the patient's abdomen.

As to the question of treatment much could be said, but I shall limit myself to the conduct to be followed in cases where the diagnosis, in the first place uncertain, may be made a sufficient probability of either appendicitis or typhoid, as well as those cases where uncertainty still remains after a careful examination of the various diagnostic elements that we have considered. The rules which should guide the practitioner may, according to our way of thinking, be laid down somewhat as follows: In the first place we will suppose that the diagnosis falters, but only as far as typhoid fever and appendicitis are concerned, and that nothing in the evolution of the disease would lead one to suspect a pathological association of the two processes. Under these circumstances the plan to follow is generally a simple matter. The clinical evolution should be carefully studied and one should surround one's self with all guarantees which are accessorily furnished by the blood, urine, and Widal's test. If the diagnosis inclines in favor of typhoid,

operative interference should naturally be deferred, at least until new symptoms appear which change the first diagnosis. If, on the contrary, the case is one of appendicitis, although one may still have some slight doubt, operation should be at once proposed in order to avoid the always menacing possibility of irremediable accidents, because it too often happens that the surgeon is called too late, but one can never intervene prematurely. However, it may happen that the case is a particularly difficult one, in which all the appearances are in favor of appendicitis, when in reality one is dealing with typhoid. Now, in this case, what will occur if laparotomy is done and the diagnostic error found after the abdomen is opened? Assuredly the operative shock, the narcosis, the pain, and all the ordinary consequences of such an interference are not to be desired in a typhoid infection, but a number of published cases would lead me to suppose that the prognosis is not bad, because the general condition of the patient has not appeared to be greatly aggravated by the operation.

There now remain those cases where during the progress of a typhoid symptoms of appendicitis make their appearance. The course to follow under these circumstances is subject to much discussion, and without considering all that has been said upon it we will simply consider two phases of the question which, according to our way of thinking, are most dissimilar. In the first case we have typhoid symptoms dominating the scene, while those arising from the inflammatory process in the appendix are, or appear to be, of secondary importance. It is possible, as I have already pointed out, that in these cases the appendix is the seat of only a temporary hyperæmia, which, as is known, is not an infrequent occurrence during typhoid. Here the prognosis is relatively favorable and an operation will probably not be necessary. But when the appendicular symptoms take on an intensity capable of leading one to suspect an ulcerative process in the appendix, or when the symptoms of appendicular reaction, in the first place slight, become aggravated, operation is indicated without delay. In closing, I think I can sum up in a word all that has been said in this paper, namely, that every time an appendicitis exists, either alone or associated with typhoid, an operation is indicated. Interference is contraindicated only in cases of simple typhoid, or one associated with symptoms arising from the appendix, but which are so slight that they cannot be considered as due to a true case of appendicitis.

THE LAW AND THE DOCTOR.[1]

By C. Stuart Patterson, Esq.,
of Philadelphia.

I have selected as the subject of my remarks this evening, "The Law and the Doctor." Under that title I shall, in general terms, discuss the various relations with the law into which the doctor is brought by reason of his profession and his practice; but I shall not attempt the impossible task of providing you with a guide to take you safely through all the mazes and the intricacies of the law.

Under our complicated form of government there are, as you know, laws of the United States, laws of the several States, and municipal regulations of the cities and towns. With the laws of the United States you have, as practising physicians, nothing to do, unless you become medical officers of the army or navy, or Immigration and Quarantine Physicians, or Examining Surgeons under the Pension Bureau; or unless you should reside and practice in the District of Columbia, or in some other Territory directly governed by the laws of Congress. It may, however, be, under the restless activity which now dominates the government at Washington, and under the modern latitude of constitutional interpretation, that it may hereafter be determined to be interstate commerce for a doctor in Philadelphia to send a prescription to a patient in Camden, or for a patient in Wilmington to be brought to a Philadelphia Hospital for operation; or it may be determined that there ought to be one uniform regulation equally affecting medical practice in every part of the United States. Such a conclusion would not be more revolutionary than some that are now discussed. That determination has not yet been made, and doctors will probably not be brought under Federal regulation and control until after the government of the United States has succeeded in fixing the prices at which all commodities are to be sold, and in prescribing the minimum number of children permissible to any well-regulated family. The laws, therefore, which now concern you are the statute law and the common law of the State, and the police regulations of the district, city, or town, in which you may open your office and practise.

At the outset there are to be considered the legal restrictions upon the right to practice your profession. That right has been in England a subject of legal control ever since the third year of the reign of Henry VIII., but in this country the restrictions are of recent origin and development. It is obviously one of the duties of the government to protect its citizens as far as possible against incompetent practitioners, but because of the essential inadequacy of all

[1] An address delivered before the Academy of Jefferson Medical College, April 14, 1905.

human methods of testing competency, otherwise than by actual experience in practice, all that the State can do is to establish certain prerequisites, which, when complied with, shall afford a reasonable probability that the practitioner has undergone a certain minimum of preliminary training. The statutes of each State must, of course, be looked to for a full and accurate statement of the prerequisites required by that State. Speaking generally, the statutes require satisfactory proof that the applicant has attained the age of twenty-one years, is of good moral character, has a diploma from a medical school of recognized standing, given him after attendance upon a course of specified length, has passed a satisfactory examination before a State Board of Examiners, and has filed in a public office the official certificate of his compliance with these prerequisites. These statutes generally except from their operation non-resident consultants, and practitioners not maintaining offices within their boundaries. The statutes impose, as penalties for their violation, in some cases, a forfeiture of professional compensation, and, in other cases, fine and imprisonment.

Pennsylvania and some other States require the applicant to have obtained a competent common-school education. I am glad to know that Jefferson Medical College has, in its requirements for admission to study, gone farther than the statute, and imposed the conditions of either a satisfactory degree or diploma in the arts, or a certificate of successful examination by competent examiners in subjects of greater scope than those of a common-school education.

While it is desirable that everyone who enters upon the study of either law or medicine should, where his individual circumstances are favorable, bring to that study the broadening and the liberalizing influences of residence and training in a great university, and should have that academic degree which is the proof of his opportunities for general culture, it yet is not expedient that the law should make the possession of such a degree a prerequisite to the practice of a learned profession. To do so would be to close the doors of the profession to all save those whom the accidents of fortune permit to indulge themselves in the luxury of college life, and to postpone the time at which they become self-supporting. Under modern conditions of competition and specialization that time is, even if circumstances be favorable, so long deferred, that it is not wise to increase it by a hard-and-fast rule, whose unbending application will not only work great hardship in individual cases, but will possibly deprive the profession and the community of the services of some men of eminent natural ability.

The young doctor of to-day comes to the practice of his profession more generally and more thoroughly instructed than his predecessor of fifty or even twenty years ago. The study of medicine is, because of its technical terminology and because of the complexity of the human body, the most difficult of all professional studies. The

medical course has wisely been lengthened in time and broadened in scope. Yet, bearing in mind the true definition of education as a method of mental development and training, and not as a means of acquisition of information, it may be questioned whether there is or is not to-day a tendency in medical instruction to require of the student, in his days of pupilage, the study of too many topics and the attempt to master too many details. The danger in that is the making of smatterers, whereas the maxim of all educators should be "*non multa sed multum.*" Be that as it may, the learned professors in our great colleges, who are training the doctors of the future, may be trusted to check that tendency whenever it becomes certain that it produces undesirable results.

The statutes now in force deal, in general, with the practice of medicine and surgery. That means, strictly construed, the professional treatment of disease by diagnosis, advice as to diet and manner of life, the administration of drugs, and surgical operative interference. Whether the practitioner serves his patient gratuitously or charges for his services does not affect the application of the statutes. Nor, so long as the doctor uses physical means, is it material that he or she is a regular physician or is of the homœopathic or the eclectic schools, or only practices midwifery, or is a bone-setter, or is exclusively a devotee of Dr. Lorenz's methods. All are alike subject to the statutes. But there is a question whether those dangerous modern charlatans, the practitioners of treatment by faith, or of so-called Christian science, are or are not within the statutes. As to this the authorities differ. It has been held, on the one hand, that their non-use of physical means of treatment takes them out of the statute. It has been held, on the other hand, and with greater reason, that, as they do undertake to treat disease, though only by advice and by mental influence, they do practice medicine, and they do bring themselves within the statutes. It is to be hoped, in the interest of the community, that this view will ultimately prevail, and that all professional practitioners of the healing arts will be brought under the statutes, for, undoubtedly, lives have been lost which would have been saved if proper medical treatment had not been prevented by the teachings of ignorance.

The relation between the doctor and his patient is, in the highest degree, confidential. It is necessary for the professional adviser to know all that he can observe, and all that the patient can tell him, as to the cause and nature of the patient's condition, and it is essential to the patient's freedom of communication that he should feel assured that the doctor will regard as confidential all that he sees and hears. The members of the profession have always observed this honorable obligation of secrecy; but the law has not, until recently, permitted the doctor, when called as a witness, to refuse to violate the confidence reposed in him by his patient. Now, in many jurisdictions, the statute declares that "a person duly author-

ized to practice physic or surgery shall not be allowed to disclose any information which he acquired in attending a patient in a professional capacity, and which was necessary to enable him to act in that capacity." In some jurisdictions the protection is limited to other than criminal proceedings. In other jurisdictions, notably in Pennsylvania, the privilege is further limited to that "which shall tend to blacken the character of the patient." In no jurisdiction does the privilege protect consultations for the purpose of committing an act forbidden by law. In all jurisdictions, as the privilege is intended for the protection of the patient, he may waive the privilege, and, if he does so, the doctor may and must testify fully. It ought in all jurisdictions to be determined that the privilege cannot be asserted in lunacy proceedings, or in cases involving testamentary capacity, for otherwise the ends of justice might be defeated. It would be wise for the members of the profession to consider this subject and to endeavor to have the law so amended that the privilege, while more fully protecting the interests of the patient, will not in any way prevent the furtherance of justice.

The doctor is next brought into contact with the law by various regulations of police, which more or less directly affect him in his practice. For the terms and effect of and the results of disobedience to those regulations, the statutes or ordinances, putting them in force should be carefully examined. The most important of those regulations require reports to public officers of births, cases of contagious disease, and deaths, and causes of death, and regulate the distribution of the unclaimed bodies of the dead for dissection, demonstration, and study. There are also in some jurisdictions regulations as to the prescription and administration of poisons, narcotics, and dangerous stimulants. There are also, in many jurisdictions, other sanitary regulations as to which it is well for all practitioners to accurately inform themselves.

The interests of the medical profession and those interests of the community which are inseparably bound to the interests of the profession have not been, as yet, injuriously affected by governmental regulation.

There are, however, tendencies which may in the future produce undesirable results. There are in every community busybodies, who, without adequate knowledge or training, stimulate popular agitation upon technical subjects. The modern agencies of communication, the railroad, the frequent mails, the telegraph, the telephone, and the sensational newspaper, have rendered it easy to manufacture upon any subject an apparent force of public opinion, which is really nothing more than the consolidation of the ignorance and the prejudices of the uninformed, and which, nevertheless, can, and often does, effect hasty and ill-considered legislation. When unwise governmental action is threatened, it can only be met by professional organization and by a campaign of education.

The nature and the extent of the doctor's duty to his patient may be brought into discussion in legal proceedings, either where a patient becomes dissatisfied with the results of his treatment, and brings an action to recover damages for that which he alleges to have been malpractice; or where a doctor, having sued to recover his professional compensation, is met with the defence that the treatment was unskilful.

A doctor may, if he is sufficiently foolish, expressly guarantee his patient's restoration to health, or, in other words, constitute himself an insurer of the success of his treatment; but only quacks and charlatans assume any such obligation. In the absence of an express contract, the implied obligation of the doctor is that he will perform that duty of care for his patient which the law defines and requires.

It matters not whether the services are rendered gratuitously or are properly compensated, for the confidence induced by the undertaking is an adequate consideration for the implied obligation. The standard of that care is and ought to be high. The law, therefore, requires the doctor to exercise with regard to his patient, not only that degree of care which a man of ordinary prudence would exercise under the circumstances, but also that higher degree of care which ought to be exercised by a man who represents himself to be skilled in his calling, and who has assumed to do that whose right performance requires special knowledge and skill.

The doctor's lack of that knowledge, care, and skill is, if injury to his patient results therefrom, actionable negligence on his part.

A surgical operation and a physical examination involving any contact with the person are equally in law regarded as assaults, and it, therefore, is advisable that the operating surgeon or examining physician should, before the operation or examination, obtain the assent of the subject, and, if the subject be a minor of tender years, also of his or her parent or guardian. An assent to a specified operation gives no warrant for another and different operation.

Neither a husband, a parent, or a guardian can be permitted to deprive of the benefit of needed treatment a wife or a child who is of an age sufficient to give consent. While it is not legally necessary in the case of a wife or a child of such age that the consent of the husband, parent, or guardian be obtained, it is, nevertheless, wise for the doctor to secure it.

If an examination involves an exposure of the person, though without physical contact, it is advisable to obtain the like assent.

It must be borne in mind that if the operation be one which a surgeon of ordinary prudence would not undertake to perform, the assent of the patient will not constitute a defence to the surgeon, if he be sued for malpractice.

The law neither can nor does attempt to discriminate between different schools of practice, nor determine which school has the better methods. The law does, however, recognize the fact that in

all schools the practice of medicine and surgery is now highly specialized. The general practitioner is not required to be in any branch of practice all that a specialist in that branch can be. Nor is a practitioner in a country district expected to possess the knowledge or skill of one who lives in a centre of population with larger opportunities to see and to learn.

On the other hand, a specialist is held to a higher standard, and is bound to exercise that knowledge and skill that practitioners in his special line may reasonably be expected to have.

While the patient cannot be expected to have that discriminating knowledge which will enable him to determine to which class of specialists he should submit himself for treatment in every particular case, he yet is held to know certain broad distinctions. He is not entitled to insist upon homœopathic treatment from a regular physician, or *vice versa*. If he puts himself under the care of a Christian Science healer, he cannot reasonably complain if the treatment does not continue to be effective. If he consults for his own ailments one of that class of specialists who devote themselves to the treatment of the diseases of four-footed patients, he will have only himself to thank and to hold responsible if his doctor should fail to display horse-sense in his treatment. This principle is illustrated by one of the oldest-reported cases of malpractice, a Mahomedan case, where "a man, who had a disorder in his eyes, called on a farrier for a remedy; and he applied to the eyes a medicine commonly used for *his* patients; the man lost his sight, and brought an action for damages; but the judge said: 'No action lies, for if the complainant had not himself been an ass he never would have employed a farrier.'"

On the other hand, the practitioner should recognize the fact that the specialization of his profession is the necessary result of the increase of knowledge beyond the limitations of human capacity, and that it is not possible for any mortal man to know all that can at any given time be known in every branch of medicine and surgery. The doctor should, therefore, unless in a case of emergency, where the aid of a specialist is not immediately available, refuse to assume the grave responsibility of undertaking to treat that which can, with reasonable certainty of benefit to the patient, be better treated by a specialist. Under such conditions, the doctor's rashness will render him liable in damages if injury to his patient result from the treatment.

The law does not presume that every practitioner possesses the greatest possible knowledge, nor the highest possible skill. The law recognizes the fact that the accidents of fortune deny to equal merit equal opportunities to gain experience and to acquire that skill which only experience can add to knowledge; and that, given equal educational advantages, equal experience, and equal desire to become skilful, the difference in the personal equation will always

distinguish one man from another. The test is, therefore, the doctor's possession of that degree of knowledge, intelligence, carefulness, and skill which may be reasonably expected of an average practitioner of the particular school under the conditions of his environment.

The maximum of professional efficiency changes from day to day with the progress of discoveries in science, and with improvements in methods of treatment and of operation. The use of anæsthetics, general and local; antiseptic precautions in operating; the germ theory of disease; the development of sanitary science; the administration of antitoxins; the disappearance of the bleeders and cuppers; the diminutions in doses of medicines; the general employment of trained nurses; and the specialization of the profession, are a few illustrations of the changes which have come in the lifetime of men now living, and which have forced themselves upon the attention of even unobservant laymen. That system and those appliances whose use would, but a few years ago, have been regarded as a fulfilment of the doctor's duty to his patient, would to-day be universally held to fall far short of an adequate performance of that duty. No man can doubt that the future has further advances in store. But a doctor cannot be required to use immediately upon its appearance every new remedy or appliance that may be brought to his attention. The suggested improvement must have passed beyond the stage of experiment, and, even if its applicability be clear in theory, that probable sufficiency must have received the seal of an established practical efficiency before the doctor can be held to be negligent because of a failure to employ it. The test is, whether or not the particular method of treatment or operation is of recognized efficacy, and in general use by careful practitioners of the particular school under like conditions.

That rule which the law expresses in the maxim "*Respondeat Superior*" applies to medical men. Every doctor is, therefore, held to liability for the acts and omissions of his agents. The term "agents" includes assistants employed by the doctor and brought into the case by him to assist him in the treatment or operation; but it does not include consultants or specialists, practising independently, though nominated by the doctor and brought in at his instance; nor does it include those ministering angels of mercy, the trained nurses, now employed so universally and with such beneficient results; nor does it include the apothecary by whom the medicines prescribed by the doctor are supplied. The consultant, the specialist, the nurse, and the druggist are all "independent contractors," and each is held liable for his own negligence, and the doctor is not held liable for the negligence of any of them, unless it be shown that, if selected by him, he has been negligent in their selection; or, in other words, that the individual selected is so generally known to be incompetent that a person of reasonable care-

fulness would not have selected him or her. If, however, any of those "independent contractors," whether or not selected by the doctor in charge, should, subsequently to their selection, so manifest their incompetency that a doctor of reasonable skill ought to be aware of it, the doctor will be held to be negligent if he does not insist upon the removal of the incompetent.

In addition to bringing to the service of his patient his knowledge and skill, the doctor must also be as frequent and as long-continued in his attendance as the necessities of the case require; and when he ceases his visits he must give such directions as may be necessary for his patient's conduct and manner of life under the conditions of convalescence.

Any shortcoming on the doctor's part or on the part of any agent of his, in the exercise of reasonable knowledge, intelligence, and skill, in faithful attendance, in the selection or retention of independent contractors, or in necessary advice as to precautions to be taken by the patient in the future, will constitute negligence, and will subject the doctor to a possibility, not only of a forfeiture of his professional compensation, but also of a liability in damages for malpractice.

The gravamen of the action being negligence, a doctor cannot be held liable for an honest mistake in the exercise of judgment or for a failure to rise to the height of an emergency, if he has done all that a physician or surgeon of average knowledge and skill could reasonably be expected to do under the circumstances. This principle may find an illustration when, in the course of an operation for one cause, there is discovered another cause, for which further or other immediate operation may be imperatively necessary. Under those conditions the surgeon is called upon to assume a grave responsibility. The patient's consent to the primary operation does not necessarily include an implied consent to a secondary operation. Nevertheless, the surgeon must courageously do that which he believes to be best for his patient, whatever it be, and trust that the law will protect him.

In every case and at every stage of the case where a choice has to be made between courses of action, whether the subject for decision be a reference to a specialist, or the calling in of a consultant, or the continuance of undivided responsibility, or a change in the treatment, or an exploration, or an operation, either original or secondary, the doctor should ask himself what, with his knowledge of the conditions, he would, if he were the patient, insist upon. That self-inquiry will not relieve the doctor from liability, if, in his answer to it, and his consequent action, he sins against knowledge; but it will, if the question be fearlessly answered, save the doctor, in most cases, from mistakes that will be hurtful to the patient, and may be injurious to the doctor's reputation.

An action for malpractice, whether well or ill founded, is a great

injury to a professional man; and the law puts upon the patient who makes such a charge the burden of proving by a preponderance of evidence that the doctor has failed in the performance of his duty; for the presumption in the case of one who has been legally licensed to practice is that he was duly qualified, and that that which he has done was rightly done. Of course, the doctor cannot be held responsible if the patient fails to obey the doctor's directions or undertakes to act upon his, the patient's, own judgment as to the treatment.

In cases of malpractice, the damages, if any be awarded, will generally be compensatory in character, and will be measured by the injury to the plaintiff directly caused by the doctor's negligence, and resulting in bodily pain and suffering, present or prospective, in injury to health, in diminished physical or mental capacity, in loss of present or future earnings, and in actual expenditures, or legal liability incurred, for subsequent medical or surgical treatment, but not including counsel fees, nor interest upon the damages accruing prior to the verdict therefor. When the injured plaintiff is a wife or child, an action may be brought on her or his behalf, and the husband or parent may also sue on his own behalf, and recover, when the injured patient is a wife, damages for the loss of his wife's society and services; and where the injured person is a child or servant, compensation for the loss of services during the time of service or during the minority of the child.

Where the doctor's malpractice causes the death of the patient, those in whom a statute vests a right of action may recover damages based upon the material, but not the sentimental, loss to the plaintiffs caused by the death. Exemplary damages, which are damages in amount more than compensatory, will be given only where the evidence clearly shows that the doctor has acted wantonly or perversely, and with such criminal indifference to his obligations as to amount to proof of a wrongful intent.

When a doctor's treatment or his exercise of care is questioned in a legal proceeding, the propriety of that treatment and that care can only be determined by a consideration of the testimony of other practitioners as to the conformity of that treatment to established methods, and as to the sufficiency of the doctor's exercise of care under the particular conditions.

It is probable that actions for malpractice will be more frequent in the future than in the past. The increase in recent years in the number of lawyers dependent for their living upon the profits of their practice, and the consequent stress of competition, have enlarged that class of legal practitioners who, like Dodson and Fogg, of immortal memory, take cases on "spec," and charge nothing unless they make it out of the defendant, and who, too often, promote litigation, and win their cases by the careful coaching of their witnesses. Such pirates make the large employers of labor their chief victims; but they naturally are keen to discover any persons whom

they can either frighten into the payment of blackmail or from whom they can recover verdicts which, when paid, go largely into the pockets of the successful pettifogger. Therefore, the physician, and still more the surgeon, should, for his own protection, take such prudent precautions in obtaining the patient's consent, and having available such proof of the correctness of the treatment, as will guard him most effectually against a possible claim.

The doctor will be brought into contact with the criminal law if he violates any statute which authorizes an indictment and which imposes a penalty. A doctor may also be held responsible to the criminal law for the death of a patient, if that death has been directly caused by the doctor's want of ordinary professional knowledge or want of ordinary attention in his care of his patient; and, in such a case, the doctor's intention to cure his patient will not relieve him from the consequences of his foolhardy presumption. Criminal statutes are strictly construed, and their legal effect depends upon their precise terms. In a criminal case, the presumption is that the defendant is innocent until he has been proved to be guilty by evidence excluding any reasonable doubt.

No class of men render as much or as valuable gratuitous service as the members of the medical profession; yet the law sometimes is called upon to deal with the question of the doctor's compensation. The old theory was that the doctor's fee is an *honorarium*, and, if unpaid, is not a subject of collection by law. The modern theory is that the services of a doctor when charged must be paid. If the amount of that compensation has been fixed by pre-contract, that contract will be enforced. If no contract has been made, a recovery in law may be had upon the basis of that which is reasonable, taking into consideration the professional rank of the doctor, the character of the services rendered, and the amount of compensation usual in the locality for such services so rendered, but not taking into consideration the greater or less pecuniary responsibility of the patient. The proof of the reasonableness of the doctor's compensation may be made by the testimony of other doctors. The patient may resist payment, either by disputing the fact that the services sued for were rendered, or that they were of the character claimed, or by proving that there was malpractice on the part of the doctor.

It is to be noted that in many jurisdictions, in the administration of the estate of a decedent, the expenses of his last illness, including both medical attendance and nursing, are held to be preferred claims.

The doctor is also brought into touch with the law as an expert witness. He may be called to testify as to the results of an autopsy, or as to the nature and effect of an injury or a wound, or as to the sanity or insanity of a person, who is accused of crime, or is a subject of present or prospective confinement in an asylum, or as to the testamentary capacity of one who has made a will, or as to the

sufficiency of another doctor's method of treating a patient, or as to the proper amount of another doctor's professional compensation.

As long ago as 1553 Mr. Justice Saunders said: "If matters arise in our law which concern other sciences or faculties, we commonly apply for the aid of that science or faculty which it concerns;" and he added, with that diffident complacency and that shrinking modesty which from that day to this has ever distinguished judges and lawyers, "this is an honorable and commendable thing in our law, for thereby it appears that we do not despise all other sciences but our own, but we approve of them and encourage them as things worthy of commendation."

The general rule is that when in any legal proceeding there comes into question any subject which is "beyond the range of ordinary observation and intelligence," a person who is skilled in that particular subject may be summoned as a witness to inform the court and the jury as to it; and, of course, when the question is medical or surgical in character, a doctor may be called to testify.

The term "witness," in its primary meaning, is "one who sees or knows, personally," and, in its secondary meaning, "one who gives testimony or evidence."

It is clear that an expert is not a witness within that primary meaning. He has no knowledge as to the facts in controversy. On the other hand, he does know that which the judge and the jury cannot know, and that is the scientific inferences that may properly be drawn from the facts proved. In other words, the duty of the expert is to do, with regard to inferences which are "beyond the range of ordinary observation and intelligence," precisely that which the average man can do for himself as to inferences which are within that range. Properly speaking, therefore, the function of an expert is not that of a witness, but it is that of an assessor, that is, of one who sits by the judge and gives him the benefit of his advice.

It would be more in keeping with legal theory if the law, instead of permitting each party to a cause to call his expert as a witness, would authorize some competent appointing power to select an expert, to be adequately paid by the State, to sit with the judge, and, as an assessor, to impartially instruct the jury as to the technical questions in the case, in the same way that the judge instructs the jury as to the law. If that were the practice, we should avoid that suspicion of prejudicing partisanship, which is always suggested by the sight of an expert witness, known to have been called and to be compensated by one side, and to be engaged, not only to state his views on behalf of the side which has called him, but also to assist the counsel for that side in the cross-examination of the expert witnesses who are called by the other side. This plan would, however, involve certain practical difficulties. In the first place, if the appointment were made by political officers, considerations other than those of professional competency would probably deter-

mine the selection; and if the appointment were made for each case by the judges, the professional qualifications of the appointee might or might not be so generally recognized as to render the selection satisfactory to all parties to the proceeding. In the next place, there would be no possibility of appeal from the conclusions of the expert, or of correction of any erroneous views which he might express; for neither the judge at the trial, nor an appellate tribunal, would be competent to review his instructions. It is, therefore, probably safer to adhere to the present system, under which the conclusions of the expert can be tested by cross-examination, and have only such weight with the jury as their force and clearness of statement and their soundness of reasoning may secure for them.

It has sometimes been questioned whether or not a medical expert can be compelled to obey a subpœna, and for the ordinary and merely nominal fees of a witness give to the court and the party calling him the benefit of his testimony as an expert, when that testimony derives its value from an expensive education and from years of study and experience, and when his attendance in court may, possibly, lose him profitable business. The better opinion would seem to be that as the duty of a citizen requires him to obey, without cavil or question, the process of a court, any doctor, if duly summoned by service of a writ of subpœna, and by payment or tender of the regular witness fees, if he demands them, may be compelled to attend the court and to answer any questions that may be asked him; but he cannot be compelled in that manner to give any consideration to the subject matter of examination elsewhere than on the witness stand. Therefore, as it is one thing to take a horse to water and another and a different thing to make him drink, so it is one thing to bring an unwilling expert into court and another and a different thing to draw from him that clear and convincing testimony which will aid the cause of the party in whose behalf he has been summoned. No competent lawyer, therefore, would call an expert to testify without making satisfactory arrangements as to that expert's compensation.

As a doctor can be compelled by a subpœna to appear and testify, it follows that in the absence of an express contract of compensation he cannot legally recover as compensation more than the ordinary witness fees, and, therefore, it is wise for the doctor, when he is asked to serve as an expert, to have a clear understanding as to the amount of his compensation, and as to the responsibility of the party by whom payment is agreed to be made.

There are experts and experts. It is desirable, in the first place, that the doctor should not only know his subject accurately and thoroughly, but also that he should have sufficient decision of character and power of repression and of expression to enable him to meet the test of a searching cross-examination, and to make his view clear and convincing to the minds of men of not more than,

and often less than, average intelligence. He should be sparing in the use of technical terms, and when he does use them he should translate them, so that their meaning may be grasped by the court and jury. Above all, while firm and self-respecting in his manner, he should avoid all appearances of dogmatism and intolerance. If long and involved hypothetical questions are put to him, he should not answer hastily, but should insist, as is his right, upon full time to consider the question before answering, and to satisfy himself that no false analogy, nor any snare for the unwary, lurk within its many words. The doctor whose knowledge is real and who thinks clearly and talks plainly has nothing to fear from any cross-examination, however severe; and he will be treated by both the court and the counsel with the most distinguished consideration.

The doctor may also bring himself into contact with the law by certifying erroneously as to the insanity of a patient, who, upon that certificate, is committed to confinement. While a mistake in judgment or an error in diagnosis will not subject the doctor to any liability, yet a misstatement of a material fact will render the doctor liable in damages. Doctors should scrutinize with care every word of printed forms submitted in such cases for their signature; and they should carefully examine, or have a lawyer examine for them, the statute under which they are asked to certify.

From all this there follow three practical conclusions:

In the first place, it is the duty of every good citizen to obey the law, and this duty is in a high degree incumbent upon members of all learned professions whose education and training may properly be supposed to raise them above the common mass of people, and to give to them a higher sense of duty. If, therefore, there be in the law any regulation which seems to you to do injustice to the interest of your profession, or to yourself, work and agitate for the reformation of the law; but, so long as the regulation be in force, faithfully obey it.

In the second place, remember that it is as foolish for a medical man to undertake to determine for himself what the law is upon any subject as it is for a lawyer to attempt to prescribe for the bodily ills of himself or of any other person. Therefore, whenever you are in any way brought face to face with law, consult a lawyer, and do it promptly, for it is as true in law as it is in medicine, that "an ounce of prevention is," if it be not too late to prevent, "better than a pound of cure."

In the third place, as legal restrictions and professional ethics touch at many points, it is wise for every young doctor to form such relations of mutual confidence and regard with one or more of the seniors of his profession, that he can obtain for himself the great benefits of a senior's advice, when questions of professional expediency present themselves. Remember that every profession has its

traditions, in which is crystallized the wisdom of its past; and that seniors who have risen to eminence and who have that soundness of judgment which the young, however learned and able, rarely have, can best determine when it is and when it is not judicious to depart from those traditions.

And one word more: I envy you; not only because you are young and I am old, for the old always envy the young; but also, and chiefly because you are to enter upon the practice of the greatest of the professions; a profession whose members have it in their power to do more than other men in relieving human suffering and alleviating human misery; a profession whose history is a story of continued growth and progress, of brilliant discoveries in science and beneficent improvements in methods of treatment and of operation, and, in many instances, of martyrdom to duty.

I close with the expression of the hope that you will walk worthy of your high vocation, and that in the years to come you will be loyal to your Alma Mater and do honor to your profession and your College.

SOME OBSERVATIONS ON ANEURYSM AND ARTERIOSCLEROSIS.[1]

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HISTORY.

In tracing the history of the study of aneurysm, we find it divides itself into two periods. First, that in which the aneurysm was looked upon as a localized disease of the vessel; second, that in which arteriosclerosis was recognized, aneurysm being considered in association with an advanced stage of this change in the arterial wall.

The first of these periods, that of aneurysm, divides itself into two—that in which the peripheral aneurysm was recognized and treated, and that in which the deep seated, like the thoracic aneurysm, was recognized.

The steps in the development of our present knowledge of arterial disease as they are here presented will be appreciated by following these charts.

[1] The Mütter Lecture, delivered before the College of Physicians of Philadelphia.

CHART I.

Historical Chart. (Chronological.)

A. Epoch of Ancient Medicine (1500 B.C.–210 A.D.).
- Egyptian Medicine.
- Assyro-Babylonian Medicine (Ignorance and Superstition).
- Hebrew Medicine (1500–400 B.C.).
- Indian Medicine.
- Greek Medicine (400–300 B.C.)
  - Hippocrates (460–377 B.C.) } Aneurysm confused with other tumors.
  - Aristotle (384–323 B.C.) }
  - School of Alexandria (300 B.C.).
- Roman Medicine (100 B.C.–210 A.D.).
  - Asclepiades (100 B.C.).
  - Celsus (First Century).
  - Galen (131–210 A.D.). (Term "Aneurysm"—"widening." Arteries contain blood, not air.)

The epoch of ancient medicine, which may be said to extend from 1500 B.C. to 200 A.D., is represented by Egyptian, Assyro-Babylonian, Hebrew, Indian, Greek, and Roman medicine. In this epoch, doubtless, peripheral aneurysms were seen but confused with tumors of other nature. In Greek and Roman medicine occur the names of Hippocrates (460–377 B.C.), Aristotle (384–323 B.C.), Herophilus and Erasistratus of the school of Alexandria (300 B.C.), Asclepiades (100 B.C.), Celsus (first century), Aretaeus (second century), and Galen (131–210 A.D.). It is the last-named who probably makes the first definite reference in literature to the condition and who used the term *'Ανέυρυσμα*, meaning a dilatation.

It will be remembered that it was Galen who declared that the arteries contained blood not air, the latter view having been held by Erasistratus of the school of Alexandria. (Dezeimeri's *Dictionnaire Historique de la Médecine Ancienne et Moderne.*) The translation of the passage from Galen's work in which he refers to aneurysm is the following: "When the mouth of the artery is opened the affection is called *'Ανέυρυσμα* (dilitation). The same thing happens when the artery being wounded, the incumbent skin is cicatrized, but the wound of the artery remains, being neither conglutinated, covered with a cicatrix, nor shut by incarnations. Affections of this kind are known by the pulsation of the arteries, or more especially by compressing the artery, for then all the tumor disappears, the matter that caused it recurring into the artery." This was written in the latter part of the second century. (Quoted by Robert James in *A Medical Dictionary*, 1743, under "Aneurysm.")

CHART II.

Mediæval Medicine (300–1400 A.D.).
- Byzantine Medicine.
  - Oribasius (326–403 A.D.). Recognized peripheral aneurysms.
  - Paul of Ægina (625–690 A.D.). Recommended ligation of brachial artery for aneurysm at bend of elbow.
- Arabian Medicine.
  - Avicenna (980–1036 A.D.). Writes on peripheral aneurysm.

The next epoch, that of mediæval medicine, which may be said to extend from 200 to 1400 A.D., and which consists of the Byzantine (300–700) and Arabian (800–1000) periods, also the school of Salerno (1000–1200) and that of Mundino (Mundinus) (1276–1326), contains but indefinite reference to peripheral aneurysms, though in the Byzantine period, Oribasius (326–403) ligated arteries for cure of aneurysm, and Paul of Ægina (625–690) recommended ligation of the brachial artery a few fingers' breadth below the axilla for cure of aneurysm at the bend of the elbow (Hodgson), and in *Arabian Medicine* we find that Avicenna (980–1036) was familiar with peripheral aneurysms. Most authors attribute the first recognition of peripheral aneurysm to Avicenna, though from the above it is evident that these aneurysms were recognized and even treated four or five hundred years before Avicenna.

In these periods, however, all of those fundamental facts of gross anatomy and physiology were being sought for, so that beyond a few observations of historic interest these periods contain no contributions to our knowledge of arterial disease. Until anatomy was systematically studied, this whole subject remained in obscurity. The advances in anatomy, indeed, amounted to very little up to this time, the beginning of the epoch of modern medicine which begins with the time of Linacre (1461–1524) and extends to our own. Prior to this (1400) the studies of human anatomy may be estimated by the fact that Mundino (1276–1326), who was professor of anatomy at the University of Bologna during the latter part of the thirteenth century, "dissected in eleven years only three human bodies." From the period of the Alexandrian school (300 B.C.) to this time of Mundino (1300 A.D.) the human body had never been dissected (Chapman). With the beginning of the sixteenth century, peripheral aneurysms were well recognized, Paré (1517–1590) introducing the method of ligating on both sides of the aneurysm, allowing, however, the intervening area to slough out. He also definitely differentiated abscesses from aneurysms.

The discussion now turns to the possibility of aneurysms of deep-seated vessels. To Vesalius (1514–1565) is due the credit of making the first diagnosis of intrathoracic aneurysm during life. This aneurysm followed injury and was detected by examining the back of the patient; a point of great clinical importance rarely mentioned, and one alluded to in a recent paper by Osler.

"The Doctrine of Dilatation of the Aorta," however, made so slow a progress that in the year 1596 when Sylvaticus published his "Treatise on Aneurysm" he did not mention internal aneurysm, and many years afterward (1658) Riolanus asserted that "aneurysm happened but seldom in the trunk of aorta in consequence of the thickness of its parietes." Elsnerus prefixed to his observations concerning aneurysm of the arch of the aorta *De Paradoxico Aneurismate*. The *Sepulchretum Anatomicum* of Theophilus and the

Historia Anatomica Medica of Lieutaud, Morgagni characterized as "unsystematic and vague."

Of thoracic and deep-seated aneurysm Morgagni says: "From the year 1670 they ceased to be considered as rare occurrences; and we have now acquired such familiarity of the disease from its causes and symptoms that we believe it may be distinguished, even when very small, and entirely concealed within the thorax." Malpighi, who was partly a contemporary of Morgagni "accounted the knowledge of dilatation of the aorta among the improvements of (his) age."[1] About this time (1670) Valsalva (1666–1723) tied the carotid of dogs repeatedly, and in one instance he tied both carotids, the dog living twenty-two days (Hodgson).

Much valuable clinical and pathological information was added by Johannes Mariana Lancisi (1654–1720) in his *De Motu Cordis et Aneurismatibus*, and by Haller (1708–1777), who in his *Opuscula Pathologica* (vol. x. and book vii., p. 127) introduces the term "Atheroma" and demonstrates collateral circulation, also by John Hunter (1728–1793), who tied the distal end of the vessel for cure of aneurysm (1785).

In the writings of Malpighi (1628–1694), Valsalva (1666–1723), and Morgagni (1682–1771), as embodied in his *De Sedibus et Causis Morborum*, published in 1760, are systematic ante-mortem and post-mortem descriptions of aneurysm. Here we find accurate reports of aneurysm of the splenic, popliteal, and inguinal arteries. Of arteries in general we find reports on ulceration of the coats of the vessel. Of the aorta we find reports on atheromatous coats, thickening and induration, inflammation, opaque spots, ossification, callous, cartilaginous deposits, incipient aneurysm, advanced aneurysm, and ruptured aneurysm. Case 26 is the report of sudden death resulting from rupture of one of the coronary arteries.

In 1805 Sir Astley Cooper ligated the carotid artery, a step which, as already stated, had been taken by Valsalva experimenting upon dogs. In 1811 Joseph Hodgson contributed his *Treatise on the Disease of the Arteries* which contains observations upon the pathology and treatment. Antonio Scarpa (1748–1832) also published an exhaustive *Treatise on Pathology and Surgical Treatment of Aneurysm* which was translated into English from the Italian about 1830. The clinical contributions of Laennec (1781–1826) and of that great teacher Louis (1787–1872) must not be omitted. From Corvisart (1755–1821) we get the term "vegetation."

Peripheral aneurysms then were recognized and treated as far back as the third and fourth centuries. Deep-seated aneurysms were recognized about the end of the seventeenth century.

[1] Seats and Causes of Disease, xviii., 14; Cook's translation, 1824, p. 330.

CHART III.

Historical Chart. (Chronological.)

Modern Medicine (1400–1900 A.D.).
Revival of Anatomy. Vesalius (1514–1565). Thoracic aneurysm diagnosed during life.
Practical Surgery. Paré (1517–1590). Ligates on both sides of aneurysm.
Circulation. Vascular system including capillaries demonstrated.
Harvey (1578–1657). Circulation.
Malpighi (1628–1694). } Capillaries.
Van Leeuwenhoek (1632–1723). }
Gross. Pathology.
Morgagni (1682–1771) }
Malpighi (1628–1694) } Peripheral and deep-seated aneurysm and other diseases of the arterial wall recognized and described.
Valsalva (1666–1723) }
Medical, Surgical, and Pathological.
Lancisi (1654–1720). "De Motu Cordis et Aneurysmatibus."
Haller (1708–1777). Term "Atheroma."
Auenbrugger (1722–1809). Percussion.
John Hunter (1728–1793). Ties distal end for cure of aneurysm.
Astley Cooper. Ligates carotid (1805).
Scarpa (1748–1832). "Treatise on Pathology and Surgical Treatment of Aneurysm."
Joseph Hodgson. "Treatise on Pathology and Treatment of Aneurysm" (1811).
Corvisart (1755–1821). Term "Vegetation."
Laennec (1781–1826). Auscultation.
Abernethy. "Origin and Treatment of Aneurysm" (1817).

Coming more to our own times, we find the observations upon the diseased arteries more specific as indicated by such terms as "arteriosclerosis" introduced by Lobstein in 1834 and "endarteritis obliterans" by Friedländer. "Arteriocapillary fibrosis" was introduced by Gull and Sutton, who recognized the condition as an independent affection.

The first theory upon which arterial disease was studied was that of inflammation, a view held by such men as Scarpa, Haller, Kreysig, Lobstein, and Bizot. It must be remembered, however, that at the time of these observers no very clear view of the pathology of inflammation existed, and consequently one obscure subject was being explained by another, almost equally obscure.

In 1844 Rokitansky opposed the inflammation theory and offered in its place the hypothesis, that material detrimental to the tissues of the arterial wall was deposited from the blood stream. Opposed to this view were Engel, Crisp, and Neumann, while Donders and Jansen were in support of the Vienna pathologist.

The important studies from this point on, as the microscope and histological technique improved, were concerned with the minute anatomy of the bloodvessels, also the physical laws governing their degree of dilatation, elasticity and resiliency.

Resse, a strong opponent of Rokitansky, presented important histological observations in support of the theory of *interrupted nutrition* and new connective-tissue formation.

It was Virchow, however, who was the strongest opponent, and to him is attributable the complete overthrow of the humero-

pathological doctrine of Rokitansky. Virchow divided the process into two: first, a simple degeneration; second, an atheromatous degeneration. The first he considered as a passive occurrence, while the latter he looked upon as an active formative process. The hyperplasia of the intima he regarded as any other chronic inflammatory process. The sclerosis he considered to be a chronic inflammation, as had Dittrich before him, and to it he gave the name "endarteritis chronica deformans."

With the advance in the knowledge of the minute anatomy of the vessel wall, but more especially of the pathology of inflammation made in consequence of Cohnheim's and Metchnikoff's observations, new questions arose, such as, "What is the relation of the vasa vasora to the inflammatory area, and what part do the white corpuscles play in the process?" and "How do they reach the non-vascular intima directly, from the blood circulating in the lumen of the vessel or from the vasa vasora?" At or about this time much emphasis was laid upon high blood pressure as a result of continued contraction of the arterioles.

Traube was the chief supporter of this the "mechanical theory," while Rindfleisch suggested that disturbances of nutrition from slowing of the current were responsible for the disease of the wall. These were, however, only hypotheses. By careful histological search, in which they failed to find any means of communication between the non-vascular intima and vascular other coats, Götte, Koester, Stronganow, and Talma concluded that the intima became affected from the blood circulating in the lumen of the vessel. Koester, together with Friedländer, and before them Virchow, proved that cells do pass from the vasa vasora to the intima. Trompeter and Krafft, scholars of Koester, now made the following important contributions:

1. The media is always involved at or about the same time as the intima (mesarteritis).

2. That the vasa vasora play an essential part in the process of disease of the vessel wall.

3. They contributed also important anatomical facts.

Shortly after this the importance of the vasa vasora in their relation to the nutrition and disease of the vessel wall was shown by Durante's experiment.

These experiments showed that stopping the circulation of the blood in the lumen of the vessel was followed by no damage to the vessel wall, while cessation of blood flow in the vasa vasora was followed by destruction of the vessel wall.

Thus, while many isolated facts of great value had been contributed, yet no very clear knowledge of the etiology and sequence of events in this prevalent disease up to this time existed.

Thoma, as a result of his studies, presented an entirely new theory based largely upon histological and clinical observation.

Thoma's studies were on three conditions:

1. Vessels in the fœtus and newborn.
2. Vessels in the stump of an amputated limb.
3. Vessels showing disease.

In the first of these, the fœtus and newborn, he observed that the obliteration of the umbilical blood track (Nabelblutbalm) proceeds by way of a thickening of the intima.

In the second (the vessels in the amputated stump) he observed also that obliteration of the vessel took place by a thickening of the intima. He concluded that this thickening took place in order to compensate for the lessened amount of blood consequent upon the cessation of circulation through these vessels. These he called "Physiological Thickenings."

In the third, arteriosclerosis, he finds the same factors as in the physiological process, but appearing in different sequence. He distinguished two processes in the diffuse form of arteriosclerosis.

1. Primary.
2. Secondary.

In the primary he finds a yielding of the vessel from loss in elasticity with a consequent widening of the vessel and slowing of the blood current. Connective tissue develops in the subendothelial layers of the intima, a process which tends to restore the relation between the vessel and its contents. Slowing of the current may in itself disturb the relation between vessel and contents and so require a compensatory thickening of the intima.

With advancing years in an individual the slowing of the blood current is, *pari passu*, met by this compensatory thickening, and the relation between blood stream, vessel wall, cardiac potentiality and blood pressure is thus maintained. It is certainly a beautiful theory.

In the secondary variety the process begins in the small vessels. In arteriosclerosis nodosa the diseased areas are considered as localized points of elastic weakening, the compensatory process taking place at these points. The twisting of the vessel Thoma considered as points of diminished elasticity yielding to blood pressure. We, therefore, have the circumscribed or nodular form (of Councilman) and the diffuse form, for both of which we have the explanation that there is a primary weakening of the elastic coat with dilatation and compensatory thickening of the intima by development of connective tissue between the endothelium and underlying elastic tissue.

Many are the supporters of Thoma's theory, among these being von Schrötter, who, in *Nothnagel's System*, declares the explanation as final.

On the other hand, there are those who oppose the view, as Beneke and Pekelharing, for example, who differ from Thoma in considering a disproportion between pressure and tension and diminished resistance to be primary factors.

Marchand, too, considers that the many cases of circumscribed involvement of the intima with an almost normal media argue against a uniform compensatory intima thickening, and Bollinger asks, "Is not the mechanical theory too much considered?"[1]

R. F. Fuchs is perhaps the most active opponent of Thoma's theory. He considers that it is difficult to prove that an initial slowing of the blood current occurs and declares that it is unreliable to build a theory upon an unknown and undeterminable foundation.

The French writers, of whom Huchard is the most prominent, consider the process as one not confined to the vessels but a systematic condition manifesting itself in the vessel wall as in the kidney, the liver, and elsewhere.

CHART IV.

Theories. Nineteenth Century. (Not Chronological.)

I.	Inflammation theory.	Many observers.
	Pathology of Inflammation not clearly understood.	
II.	Humero-pathological theory, 1844.	Rokitansky.
III.	Pathology of inflammation.	Cohnheim.
	Better understood.	Metchnikoff.
IV.	Mechanical theory	Traube.
V.	Interrupted nutrition theory.	Rindfleisch.
	Question 1. Nutrition of vessel wall?	
	" 2. Passage of leukocytes into vessel wall from lumen of vessel or from vasa vasora?	
	" 3. Nutrition of non-vascular intima?	
VI.	Passage of leukocytes from lumen hypothesis.	Götte, Koester, Stronganow, Talma.
VII.	Nutrition from vasa vasora *only*, demonstrated.	Durante.
VIII.	Passage of leukocytes from vasa vasora to non-vascular intima, demonstrated.	Virchow, Koester.
IX.	Compensatory process and molecular theory.	Thoma.

For further details see Prof. Jones' monograph on "Arteriosclerosis," to which I am indebted for much valuable information.

CHART V.

Nomenclature. Nineteenth Century. (Not Chronological.)

Arteriosclerosis	Lobstein.
Arteritis.	
Atheroma	Haller.
Arteriocapillary Fibrosis	Gull and Sutton.
Periarteritis.	
Periarteritis Nodosa, 1866	Kussmaul.
Obliterative Endarteritis, 1876	Friedländer.
Endarteritis, Chronica Deformans	Virchow.
Endarteritis Nodosa or Conscripta Diffusa	Councilman.
Mesarteritis	Trompeter-Krafft.
Angiosclerosis	Thoma.

Subdivisions of aneurysms not given, as these refer more especially to gross than to histological appearances.

[1] Allbutt, vol. vii. p. 303.

CHART VI.

Epochs.

Prior to 400 B. C. . .	Complete ignorance.
400 B. C. to 300 A. D. .	Beginning of accurate observation. ANATOMICAL FACTS.
300 to 1500 . . .	Decline of Anatomic study. Superstition and prejudice.
1500 to 1600 . . .	Revival of ANATOMY. Anatomical Laboratories.
1600 to 1700 . .	PATHOLOGY. Pathology (Gross). Pathological Laboratories. SYMPTOMATOLOGY and Morbid Pathology.
1700 to 1800 . . .	Methods of PHYSICAL EXAMINATION. CLINICAL.
1800 to 1900 . . .	PATHOLOGICAL HISTOLOGY. BACTERIOLOGY. PHYSIOLOGICAL CHEMISTRY.

Each of the above represents the dominant consideration of its time.

Such, then, is the history of the work done upon the arteries from the earliest epochs of medicine to our own times. It represents a vast expenditure of thought by the very ablest scientific thinkers, especially of the last three centuries.

There yet remain unanswered many questions, from among which the following have been selected, with the hope that some facts may be presented which may prove suggestive to other observers, stimulating them to investigate this widespread and important disease.

The questions are:

1. Arterial disease in animals.
2. The earliest age in which arterial disease occurs in man, and at what age it is most common.
3. The teratological factor.
4. The relation of syphilis and pulmonary tuberculosis to aneurysm.
5. Relative frequency of aneurysm in the white to colored races.
6. Relation of arteriosclerosis to aneurysm.
7. The physical properties of the arterial wall.
8. The nature of the change in the elastic tissue, which is conceded to be the most important in arterial disease.

ARTERIAL DISEASE IN ANIMALS.

The determination of the presence or absence of arterial disease in animals presents many points of interest, such as the influence of strain, as in the beasts of burden; the influence of disease peculiar to man, as syphilis; the relative elasticity of the animal's vessel and that of man, etc.

The animal's vessel possesses a greater degree of elasticity, and this may enable the vessel to withstand strain for a longer time without yielding. This offers a fruitful field for work. Horses, and more especially asses, are subject to aneurysm of the superior mesenteric and aorta due to a parasite—the *Strongylus Armatus*.

Many authors state that peripheral aneurysm is practically unknown in domestic animals.

Pigs and dogs are stated to be occasionally the subjects of aortic aneurysm.

I examined the aortas of 103 domestic animals without finding any evidence, *in the gross*, of arterial disease.

In the aorta of the steer there occurs just beyond the first portion of the arch a thickening which is, however, not pathological. This is occasionally found in man.

EARLIEST AGE AT WHICH ARTERIAL DISEASE OCCURS IN MAN AND THAT AT WHICH IT IS COMMONEST.

It is generally stated that arterial disease in children is rare, but no definite age limit is given. Thus, Keating says: "Endarteritis with degenerative changes seems to be a rare affection in children; it would appear that the bloodvessels of the brain, and the walls of the aorta are the points chiefly affected, although other large vessels are occasionally the seat of atheroma." (*Cyclopedia of the Diseases of Children*, Keating, vol. ii. p. 874.)

Schmidt, however, reports a radial aneurysm in an infant eight weeks old; Syme, a popliteal aneurysm in a boy, aged seven years, and Hodgson an aneurysm of the carotid in a girl, aged ten years.

R. W. Parker says: "After careful search of the literature," he found only 15 cases of aneurysm in those under twenty years.

Through the courtesy of Dr. Mathias Nicoll, I was enabled to analyze 799 autopsies of children who had died at the New York Foundling Asylum, the ages varing as shown on this chart.

CHART VII.

Age Chart.

Analysis of 799 Cases. New York Foundling Asylum, 1898-1903.

Stillborn to 3 months	85 cases.
3 months to 6 "	140 "
6 months to 9 "	155 "
9 months to 1 year	140 "
1 year to 2 years	141 "
2 years to 3 "	87 "
3 years to 4 "	37 "
4 years, 13 days 4 years, 1 month, 10 days 4 years, 2 months, 4 " 4 years, 7 months, 24 " 4 years, 8 months, 12 " 4 years, 9 months, 6 " 5 years, 5 days. 5 years, 14 " 5 years, 3 months, 18 days 6 years, 1 month, 13 " 7 years, 4 months, 19 " 10 years,	1 case each.
Result of analysis:	
Arteriosclerosis	1 case.
Endocarditis	1 "
Aneurysm	0 "

This list comprises tuberculosis in many localities. Pneumonia, empyema, gangrene of lung, scarlet fever, meningitis (all types), septicæmia, abscesses, nephritis, besides many other conditions which have not the bearing upon arterial disease that the above have. In addition to these there were 10 cases of congenital syphilis.

In all 799 cases there occurs only 1 case of atheroma and 1 case of endocarditis. No case of aneurysm.

This 1 case of atheroma was that of a boy, aged two years, six months and six days, who died of bronchopneumonia. There was an old appendicitis, focal necrosis in the liver and "slight atheroma of the aorta." Undoubtedly a case of sepsis.

In order to determine the age at which aneurysm is the commonest, 340 autopsy reports from literature were analyzed, with results as shown on this chart.

CHART VIII.

Aneurysm Cases from Literature.

1 to 5 years	. . . . .	2 cases.	2¼ yrs. and 5 yrs
6 " 10 "	. . . . .	3 "	
11 " 20 "	. . . . .	27 "	
21 " 30 "	. . . . .	54 "	
31 " 40 "	. . . . .	102 "	
41 " 50 "	. . . . .	90 "	
51 " 60 "	. . . . .	48 "	
61 " 70 "	. . . . .	15 "	
71 " 80 "	. . . . .	3 "	
81 " 90 "	. . . . .	0 "	
91 " 100 "	. . . . .	0 "	
		340 "	

In regard to sex : of 347 autopsy analyses the following was found :

Aneurysm.	Male	280
	Female	67
	Total	347

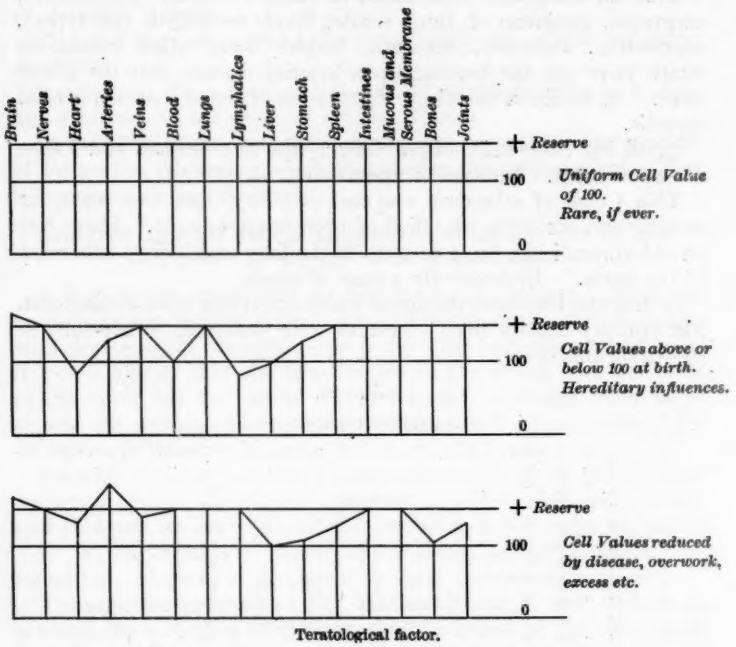
THE TERATOLOGICAL FACTOR.

A factor in arterial disease impossible to determine experimentally, but one none the less important, is that of teratology. The various parts of the body must be considered to possess a limited degree of reconstructive power.

Regeneration must have its limit. Cells of the kidney and liver, the heart, the brain, may not all have the same reconstructive energy. They differ from one another in an individual, and they differ from each other in different individuals. This is diagrammatically shown in Chart IX.

We might then speak of the "cell equation" of an individual, by which would be meant the perfection of the synergy (συν-ἔργον) of that system; thus the perfectly co-operative system would have a value of 100.

CHART IX.



Individuals rarely, if ever, start with such a value. The demands during life too soon become greater on one organ than on another, so that this inequality becomes greater. A process of compensation is at work throughout the system struggling to approximate the perfect cell energy value of 100. In this work of compensation the vascular system has a great part. It, too, however, has its limitation.

SYPHILIS AND TUBERCULOSIS.

A question, the answer to which seems to be considerably confused, is what association is there between—

1. Aneurysm or arterial disease and syphilis; also what association there is between—

2. Aneurysm and pulmonary tuberculosis.

In the question of syphilis, for example, we find Schmidt places the figures at 29 per cent., Drummond at 100 per cent., while Gerhardt finds syphilis in 53 per cent. of the cases of aneurysm. Malmesten, on the other hand, says 80 per cent. Davidson, in the *Transactions of the Pathological Society of London*, states that in 114 autopsies he found 78 cases without syphilis, in 4 of which there was atheroma of the aorta; 28 cases with syphilis, 17 of which had

atheroma—that is, 68 per cent. without syphilis, of which 5 per cent. had "atheroma," and 40 per cent. with syphilis, of which 60 per cent. had "atheroma." These are widely differing figures which probably arise from the great prevalence of syphilis. Had we a specific virus or organism of syphilis, as in the case of typhoid, cause and effect could be more certainly determined. I approached the question by analyzing 370 reports of autopsies on aneurysm cases.

In 106 of these syphilis was definitely mentioned. In 34 it was present; in 72 it was absent. This gives 32 per cent., or about that of Schmidt.

It would appear from these figures that syphilis is an important but by no means essential factor, nor is it invariably followed by disease of the vessel. In this question the age at which arterial disease is found must, of course, be taken into consideration.

In this connection Allbutt says: "I am beginning to regard atheroma of the aorta in young people as strong presumptive evidence of syphilis,"[1] though he regards syphilitic endarteritis as a distinctive process from atheroma from other causes.

I attempted to determine, if by present improved methods of treatment of syphilis, aneurysm was less common to-day than say fifty years ago, but reliable records on this point were difficult to obtain.

In regard to pulmonary tuberculosis, in 370 autopsies on aneurysm the lungs were examined and reported on in 98; 16 of these had tuberculous involvement of the lung, or 16 per cent.

Cases of pleural adhesions were not included in this estimate.

ARTERIAL DISEASE IN THE COLORED RACE.

Through the courtesy of Drs. Mathias Nicoll, L. Fougers Bishop, and B. T. Tilton, I was enabled to analyze 163 autopsy reports of the Lincoln Hospital, a hospital for the colored.[2] I was able to compare these, through the kindness of Dr. H. A. Christian, with 1928 autopsy reports of the Boston City Hospital, a hospital for the white. The result of these analyses is as follows:

Lincoln Hospital (colored).			Boston City Hospital (white).
Analysis of 163 autopsies.			1928 cases.
8 aneurysms, $4^{9}/_{10}$ per cent.			36 aneurysms, $1^{9}/_{10}$ per cent.
Thoracic,	6	8	
Abdomen,	1		
Iliac,	1		

Professors Osler and Welch gave me the opportunity to consult the clinical and pathological records of the Johns Hopkins Hospital. In the analysis of 1800 records the following data were obtained:

[1] Allbutt, vol. vii. p. 309.

[2] While this hospital has recently admitted white patients to its wards, the records of autopsies on the colored patients only are included in the above.

Johns Hopkins Hospital.		Undetermined.	Total.
Colored wards	22 cases	7 cases.	52 cases.
White "	23 "		

Thus 52 aneurysms were found in 1800 autopsies. The number of white patients in this hospital outnumber the colored by about 4 to 1. These figures would indicate that aneurysm was found three or four times more frequently in the colored than in the white. Taking then the combined figures of these three large hospitals, the Lincoln, the Boston City, and the Johns Hopkins, we find aneurysm from three to four times as common in the colored as in the white.

Granting syphilis and exposure to be etiological factors, the greater frequency of aneurysm in the colored is to be expected. No reliable figures on the relative frequency of syphilis in the white and colored from which to draw conclusions on that point could be obtained.

ARTERIOSCLEROSIS AND ANEURYSM.

A question of great clinical value is whether general arteriosclerosis is or is not an indication of tendency to aneurysm.

This question suggested itself by the fact that cases of aneurysm of the aorta not infrequently have soft radials.

In pursuit of this point, 370 autopsy reports of aneurysm cases were analyzed, in 300 of which no reference was made to the peripheral vessels. In 70, however, the vessels were carefully examined throughout; 6 of these showed general arteriosclerosis; 64 of these showed no arteriosclerosis.

When the vasa vasora are involved first, localized softening of the media occurs with secondary changes in the intima. This weakening of the media leads to aneurysm. The intima in small aneurysms (sacculated aneurysm) we know is often not ruptured.

In arteriosclerosis, on the other hand, the intima is first involved, the media showing general loss of elasticity. General arteriosclerosis is not, therefore, necessarily associated with aneurysm.

An attempt was made to determine the relative frequency of arteriosclerosis and aneurysm in the various arteries.

Chart X. represents the result of this effort. In interpreting this chart it must be remembered that while multiple aneurysm is the exception, multiple arteriosclerosis (that is, several arteries involved at the same time) is common. Thus, of 100 sclerosed vessels, the radial will be found involved in 87 per cent., while of 100 aneurysms not one of the radial will be found.

It seems reasonable to conclude that in cases of generally thickened vessels, aneurysm is not a likely complication—*i. e.*, from the table here given it occurs in about 8 per cent.

Distribution of Arteriosclerosis. The factors which determine the distribution of sclerosis of the vessels are varied. It may,

however, be stated generally that when the peripheral vessels are markedly sclerosed the abdominal vessels are free from such involvement, and *vice versa*. Great functional demand upon any part is apt to lead to sclerosis of the vessels of that organ. Thus the cerebral vessels in brain-workers are often sclerosed. The coronaries in hypertrophied heart, the radials in laborers, etc.

CHART X.

Location Chart.

	Aneurysm (from Literature).	Arteriosclerosis (Thoma and Bregmann.)
Ulnar	½ per cent.	94 per cent.
Anterior tibial	¼ "	93 "
Subclavian	5 "	88 "
Cerebral { anterior, middle, posterior, ant. common, post. common, basilar, and mid. meningeal }	8 "	87 "
Internal carotid	1 "	87 "
Radial	0 "	86 "
Splenic	1 "	82 "
Popliteal	14 "	79 "
External carotid	¼ "	78 "
Axillary	1 "	71 "
Femoral	5 "	69 "
Common carotid	1 "	68 "
Ascending aorta	34 "	67 "
Abdominal aorta	8 "	64 "
External iliac	1 "	58 "
Brachial	2 "	55 "
Common iliac	1 "	
Innominate	3 "	
Thyroid axis	¼ "	
Pulmonary	3 "	
Coronary	1 "	
Cœliac axis	¼ "	
Sylvian	¼ "	
Basilic	¼ "	
Hepatic	2 "	
Plantar	½ "	
Profunda	¼ "	
Superior mesenteric	1 "	
Pons varolii	¼ "	

PHYSICAL PROPERTY OF ARTERIES.

What, now, are the properties of this wall whose chief element in construction is elastic tissue? The analogy between the elasticity of vessels and other elastic bodies at once suggests itself. It is found, however, by Wertheim and Weber, that the laws governing the elasticity of organized bodies, such as the vessel wall, differ from unorganized bodies, such as rubber. This difference is put down to the intrinsic moisture of organized bodies.

We have then to consider the organized structure apart from the unorganized, and must take into account four properties: elasticity, resiliency, extensibility, and resistance.

The elasticity of a substance refers (1) both to its ability to resist deformation and (2) to its ability after deformation to resume its original shape. These two ideas may be conveniently combined by supposing that no force is applied sufficient to permanently deform the object tested. Supposition number two above is, therefore, always understood to control the test; that is, that the object is not permanently deformed by the test.

The force at which permanent deformation occurs is the limit of the forces producing temporary only deformation, and this force is called the *elastic limit.*

For example, suppose that we stretch a wire or a rubber band a little, and then relax the stress. The elongation produced at first disappears when the stress is relaxed. We say that the recoil is equal to the elongation. Suppose that we then continue this process of alternately pulling and relaxing, measuring the elongations and the recoils, and increasing the pull every time. Up to a certain pull, or tensile stress, we find the recoils always equal to the preceding elongations; that is, that the deformation produced by the pull is not permanent.

After this limit (the elastic limit) has been passed we find the recoils are not as great as the preceding elongations, or that the object has stretched.

As a rule, the usefulness of anything that is permanently stretched is gone, therefore we study the properties of materials only below the elastic limit. These properties are the elastic properties.

The main elastic properties are:

1. The value of the elastic limit—*e. g.*, the pull in pounds per square inch that will permanently stretch the object. This has been defined above.

2. The rate at which the object stretches; that is, the extent of its elongation for a given pull. In a spiral spring this might be called the stiffness of the spring. Scientifically this is called the coefficient or modulus of the object's elasticity. This is a long and often confusing term, but the real idea it expresses is a very simple one. It is an application of the principle that "science is measurement." In other words, the coefficient is a numerical term by which the stiffness of objects may be compared; that is, of two objects stretching unequally under a given pull, the stiffest is that which stretches *least.* But if we measured the elasticity (that is, the coefficient of elasticity) by the stretch alone, the stiffest object would have the least numerical expression for its coefficient, which would be confusing.

For example: The spiral spring *A* under a pull of 100 pounds stretches one inch. The spiral spring *B* under a pull of 100 pounds stretches two inches. It is evident that *A* is the stiffer of the two springs, and that its coefficient is double that of *B*, yet its stretch is only one-half as much. So we say that the coefficient is measured

by the pull divided by the stretch. Thus the coefficient of spring *A* is 100 pounds, and the coefficient of spring *B* is fifty pounds.

NOTE.—It is assumed that all other circumstances of the experiment are identical in the two objects *A* and *B*. It is apparent that while *A* is stiffer than *B*, yet it may be made, say of a different metal, so that *B* may have a higher elastic limit, although its coefficient may be lower.

Chart XI., Figs. 1 and 2, presents simple diagrams which will illustrate this.

Suppose that a pull of 100 pounds elongates the spring one-half inch with a recoil of the same extent. In the diagram Fig. 1 we illustrate this by the dot *a*. Similarly we find *b* and *c*. But when we make the pull 400 pounds we get the dot *d* with a recoil from *d* to the one-and-a-half-inch mark.

Let us join the points by a line as in Fig. 2. Then this line expresses all the elasticity characteristics of the object tested. The elastic limit is 300 pounds, for after this pull the extensions increase more rapidly than the recoils, or the spring has been permanently stretched—*e. g.*, two inches from 0 to 2. The back of the curve, like that of the resistance of the spring is broken at the "hump."

Chart XI., Figs. 3 and 4, shows another diagram containing the above figures, and the similar "strain diagram" of another spring.

In this case (Fig. 3), as in the hypothetical case, *A* is stiffer (or has a higher coefficient) than *B*, yet its elastic limit is only two-thirds that of *B*.

These diagrams represent the behavior of unorganized bodies. Let us now see how organized bodies behave under similar treatment.

As a rule, the strain diagram shows a straight line below the elastic limit; that is, for the portion of it from 0 to the "hump," so that the elasticity of the springs *A* and *B* may be compared by dividing the one pull—200 pounds which they have in common *below* the elastic limit—by the corresponding extensions, viz., one-half inch for *A* and one inch for *B*, making *A* twice as stiff (or elastic) as *B*, as before assumed. In these cases the coefficient for each spring is constant, though the different springs have different coefficients. Sometimes, however, the strain diagram is represented by a curve, as in Fig. 4. In this case we suppose that the curve is straight at the point in question as at *a*, *b*, *c*, or *d*. At these points draw tangents to the curve. We then compare the inclinations of the tangents. In this case the curve *C* shows that the stiffness is diminishing, and in curve *D* that it is increasing.

This latter increasing coefficient expresses the behavior of the artery as distinguished from the decreasing coefficient of unorganized bodies.

These diagrams are not fanciful, for the specimen may be made to write them autographically.

CHART XI.

FIG. 1.

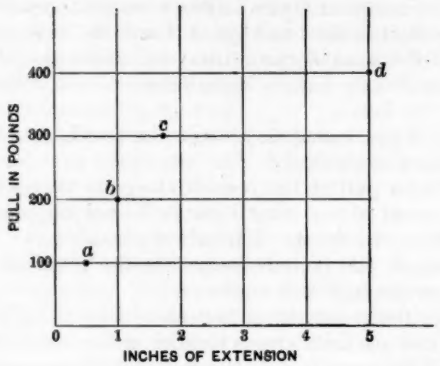


FIG. 2.

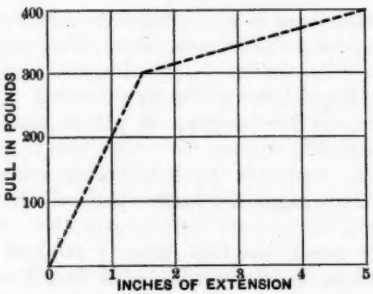


FIG. 3.

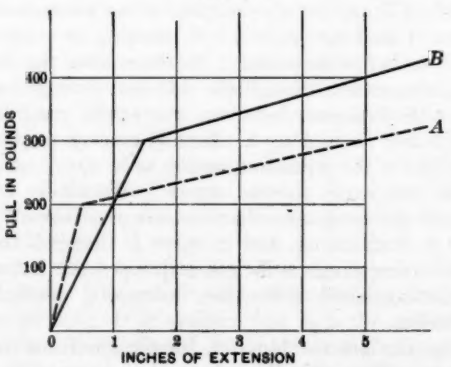
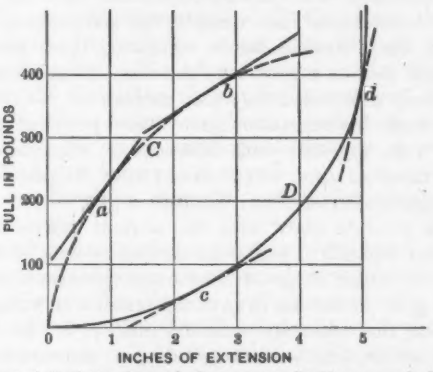


FIG. 4.



Coefficient of elasticity of organized as compared with unorganized bodies.

The elasticity of various organized substances is shown in Chart XII., also the relative elasticity of normal and diseased arteries.

CHART XII.

Relative elasticity of various tissues. (From Schäfer.)

Bones, dried	Similar to inorganic bodies.
Sinews, dried	Similar to fresh bone.
Nerves, dried	Similar to fresh sinew.
Muscles.	
Arteries.	

Relative elasticity of normal and diseased arteries:

Normal.	Weight, 100 grm.	Sclerosed.
Stretched.	Length.	Stretched.
16.5 cm.	7.5 cm.	9.9 cm.
Recovery:		Recovery:
8 cm.		8 cm.

Elasticity and resiliency diminished, especially elasticity.

To attain the greatest strength with a given amount of material, a cylinder should be constructed of layers, the elastic coefficient of which gradually increases from within outward. Now, this is exactly the arrangement of the different layers of the arterial wall; thus the greatest strength is attained by the smallest amount of material consistent with safety. Similar reasoning applies to the walls of a sphere, to which an aneurysm is comparable, from which it appears the main source of weakness in an aneurysm is, that the wall practically is composed of only one coat (Nicolls and Schäfer, p. 74). The human artery from this arrangement is physically perfect for the demands made upon it. This adaptability is demonstrated in Chart XI., Fig. 4, in which, according to Wertheim (as quoted by Schäfer, p. 72), the coefficient of elasticity of an excised strip of an artery is not constant but *with the extension increases.*

This property of increased coefficient of elasticity leads to a storing up of intermittent energy of the heart (Nicolls and Schäfer), which, if lost by disease, must greatly influence the rate of flow and thus furnish one of Thoma's essential factors in intima change.

In this connection, Dr. George McClellan calls my attention to an important anatomical fact, namely, the attachments of the great vessels to the deep cervical fascia, rendering them less movable, a fact which may be one reason why the arch of the aorta is so much more commonly affected than other arteries.

Very thorough investigation upon these physical properties of the arterial wall, together with histological observation upon the vessels experimented upon was carried out by August Luck in 1889, under the supervision of Prof. Thoma.

This work went to show that the normal artery has a greater resiliency than elasticity, while the reverse is true of the sclerosed vessel. In the initial stage of arteriosclerosis, that is, just before the thickening of the intima began, the vessel was found to be more expansile, but the elasticity was diminished. This change was found in vessels in which, histologically, no abnormal tissue could be detected.

CHART XIII.

Breaking strain of arteries.

(From various sources, together with personal observation.)

I. Normal vessels.	Pressures.
Carotid of goat.	2250 mm. Hg. 14 times the normal.
Carotid of dog.	3000 to 8500 mm. Hg.
Carotid of dog injured, intima and media slightly damaged.	At 450 mm. a pouch formed, but vessel did not rupture.
Human carotid.	1290 mm. Hg.
Large arteries rupture at lower tension than small arteries.	
II. Diseased.	
Localized sclerosis.	280 to 300 mm. Hg. Intima and media burst. Mercury trickled through adventitia.
Atheromatous plaques.	150 mm. Hg.
Generally thickened vessels.	1200 mm. Hg.

Breaking strain of veins.

Higher pressure required to rupture corresponding veins.

I am engaged in experiments along this line which seems a neglected but very important branch of the subject.

I am indebted to Captain Metcalfe, U. S. A., retired, and formerly instructor in ordnance and gunnery at West Point, for practical suggestions in the construction of apparatus to test the physical properties of arteries and for the explanation of the physical properties of unorganized substances.

HISTOLOGICAL PROPERTY OF ARTERIES.

All the evidence at hand goes to show that the seat of the most important change is the elastic tissue of the media. What is the nature of this change? It may be molecular, that is, purely physical; or it may be histological, that is, some tissue change. If the former, it can be detected only through changes in the physical properties

of the artery and microscopic appearances characteristic of molecular change in the elastic contents. If the latter, that is, histological, we should be able to detect this through staining and chemical peculiarities. That the diseased artery shows marked variation from the normal in its physical properties, the facts just stated, together with evidence not here quoted, goes to show. The question then is, are there histological changes as well, or is it solely a molecular or physical change?

Attention was therefore directed to the microscopic appearance of the elastic tissue.

Thirty-three human arteries, consisting of the aorta, the popliteal, and coronary were subjected to stains selective of the elastic tissue. These vessels were both normal and diseased—the latter being composed of all stages of sclerosis to actual aneurysm formation. About 150 sections were thus treated and compared.

The stains employed were:

1. The Weigert.
2. The Unna Taenzer.
3. The Mallory.
4. Eosin hæmatoxylin stain was employed when information upon the vasa vasora was desired.

Each specimen was stained by all of these methods, so that they present a thorough demonstration of the relative merits of each method. A great variety of fixation agents was employed and experimented with. The best fixation agent is the Müller formalin, and the best stain the Weigert.

The steps in the staining are here briefly given.

Technique Employed in Staining Elastic Tissue. After experimenting with the various fixatives and hardening agents, as well as the different methods given in works on pathological technique for staining elastic fibres, it was found that the following gave best results:

I. *Weigert's Method* (Fig. 1). *Centralblatt für Allgemeine Path. u. Path. Anatomie,* May, 1898, vol. ix. p. 289.

Tissue stained equally well when fixed and hardened in Müller, formol or Müller-formol.

Formula is:

Fuchsin (not Rubin S)	1 per cent.
Resorcin	2 "
Water	200 c.c.

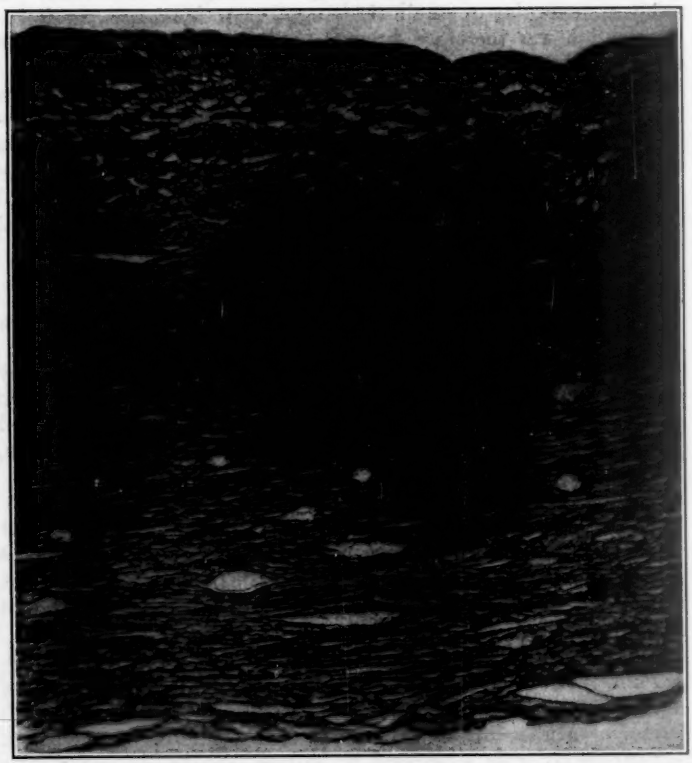
Boil in a porcelain capsule. When boiling add 25 c.c. liq. ferri sesquichlorati (Ph. G. III.), and boil 2 to 5 minutes longer, stirring meanwhile. Cool and filter.

Throw away filtrate. When precipitate is dry, place it in same capsule and add 200 c.c. 94 per cent. alcohol. Stir and let it boil, fishing out the pieces of paper. Then cool, filter, and to filtrate add enough 94 per cent. alcohol to make 200 c.c. Lastly add 4 c.c. HCl.

Stain may be used repeatedly.

For the present work, tissue was embedded in paraffin and sections cut 8 to 10μ. It was found that the best stains were obtained in ten to fifteen minutes instead of twenty minutes to one hour, as advised by Weigert, then washed in 95 per cent. alcohol, left for a few seconds in 100 per cent., cleared in xylol and mounted

FIG. 1.



Selective elastic-tissue staining. × 160. Aorta (human). Weigert. See text, p. 865. Note that the darker area in the middle of the section is due to a folding of the tissue and not to any histological change. (From the Cornell University Photomicrographic Laboratory, N. Y.)

in balsam. If overstained, acidulated alcohol hastens removal of excess.

Elastic fibres are dark blue against a pale or colorless background.

II. *Unna Taenzer, modified* (Fig. 2).

Müller, formol, Müller-formol, alcohol, and bichloride may be used for this method.

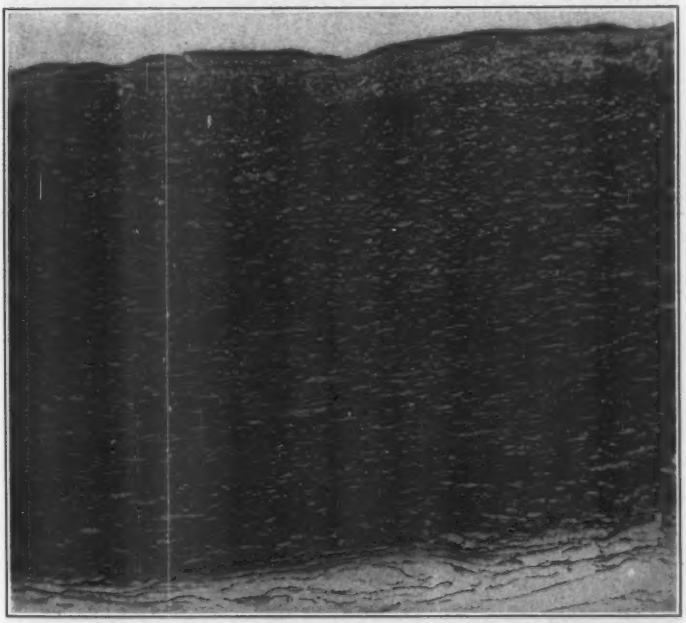
Formula for stain is:

Orcein (Grübler)	0.59 gm.
Absolute alcohol	40 c.c.
Distilled water	20 "
HCl	15 to 20 gtt.

Allow section to remain in this overnight, or about fifteen hours, then differentiate in:

Alcohol (95 per cent.)	20 c.c.
Distilled water	5 "
HCl	1 to 2 gtt.

FIG. 2.



Selective elastic-tissue staining. × 185. Unna Taenzer. See text, p. 866. (From the Cornell University Photomicrographic Laboratory, N. Y.)

Pass through absolute alcohol and mount in Canada balsam. Elastic fibres are brown against a very pale or colorless background.

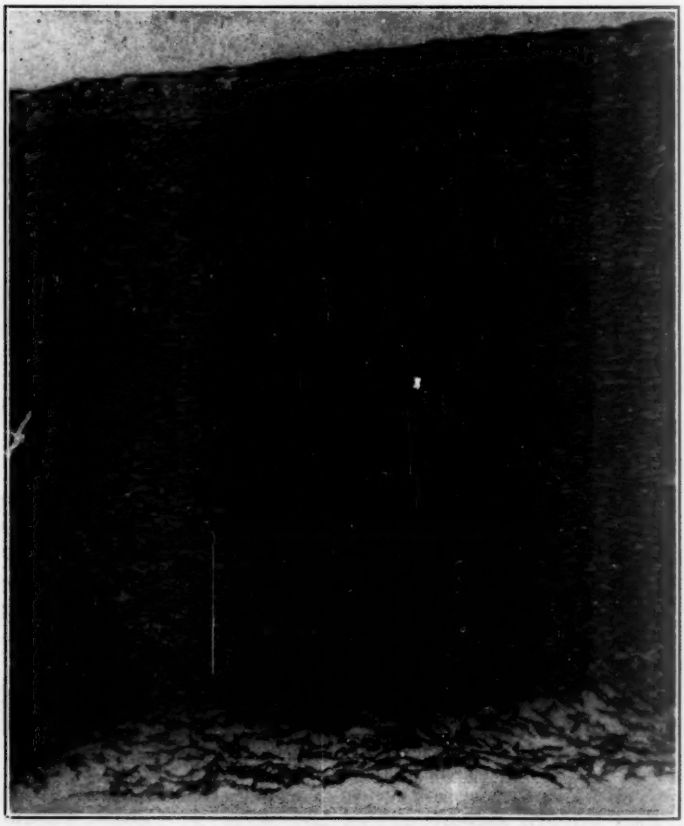
III. *Mallory* (Mallory and Wright's *Path. Technic*, p. 243, ed. 1897.)

Alcohol is the best hardening agent, a point emphasized by Mallory.

Phosphotungstic acid (1 per cent. aq. sol.)	100 c.c.
Hæmatoxylin	1 gm.

Sections are allowed to remain in stain overnight, washed in 95 per cent. alcohol, then 100 per cent., cleared in xylol and mounted in Canada balsam. If overstained acidulated alcohol will remove the excess. Fibres are a decided blue in a very pale pink background.

FIG. 3.



Tissue digestion experiments. Slightly sclerosed. × 160. Section of aorta (human) treated with 1 per cent. acetic acid. Stained with elastic-tissue selective stain. No change in tissue noted. See text, p. 867. (From the Cornell University Photomicrographic Laboratory, N. Y.)

The specimens, photomicrographs of which are here given (Figs. 1 and 2), show the following:

1. Vessels which in the gross display no signs of sclerotic change may, under the microscope, show slight thickening of the intima.

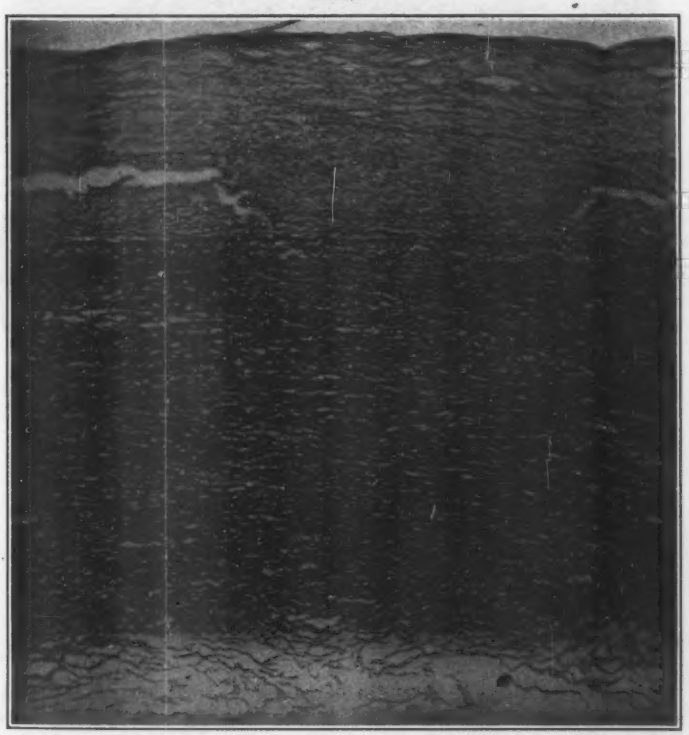
2. The elastic tissue shows the corkscrew character modified—the strands being straighter, more separated from one another, and in some instances fewer.

These then are probably mechanical changes, the stains failing to demonstrate any histological change in the elastic tissue.

DIGESTION EXPERIMENTS.

The next set of experiments was of a chemical nature, dealing with the reaction of the elastic tissue to digestants.

FIG. 4.



Tissue digestion experiments. × 185. More advanced sclerosis than preceding. Section of aorta (human) treated with 1 per cent. acetic acid. Stained with elastic-tissue selective stain. No change in tissue noted. See text, p. 866. (From the Cornell University Photomicrographic Laboratory, N. Y.)

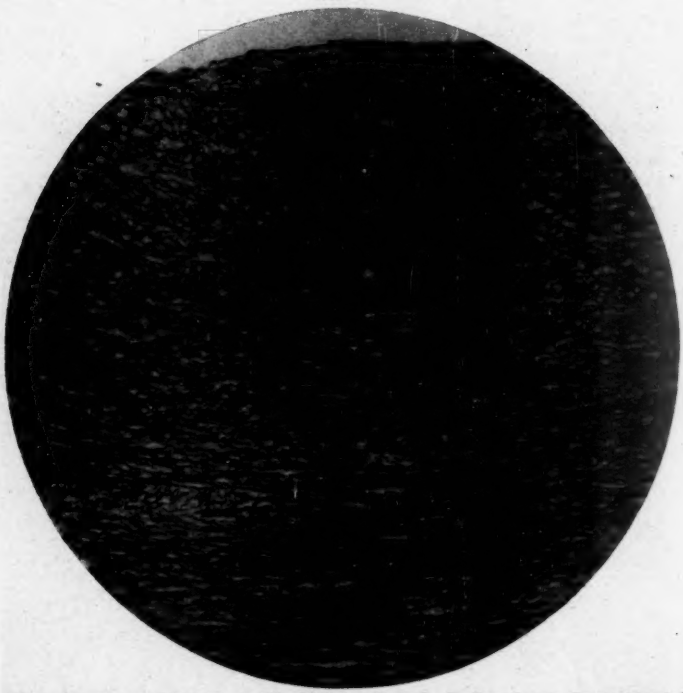
The object of this work was to demonstrate, if possible, through the action of digestants whether the change which takes place in the elastic tissue is of an histological or physical character.

The following experiments were therefore taken up. The digestants selected were acetic acid and potassium hydroxide.

Technique in the Attempt to Digest Elastic Tissue in Arteries with Acetic Acid and Potassium Hydroxide.

1. An aorta *hardened for several weeks* in Müller's fluid was chosen, and the area showing arteriosclerosis was subjected to the acid and alkali, as follows:

FIG. 5.



Tissue digestion experiments. × 185. Slightly sclerosed. Section of aorta (human) treated with 5 per cent. acetic acid. Stained with elastic-tissue selective stain. No change in tissue noted. See text, p. 866.

(*a*) *Acetic Acid.* 1. Strength of 1, 2, 3, 4, 5, 10 and 20 per cent. *acetic acid* were allowed to act in the incubator for twenty-four hours. Tissue being macroscopically unchanged, it was washed in running water for twenty-four hours.

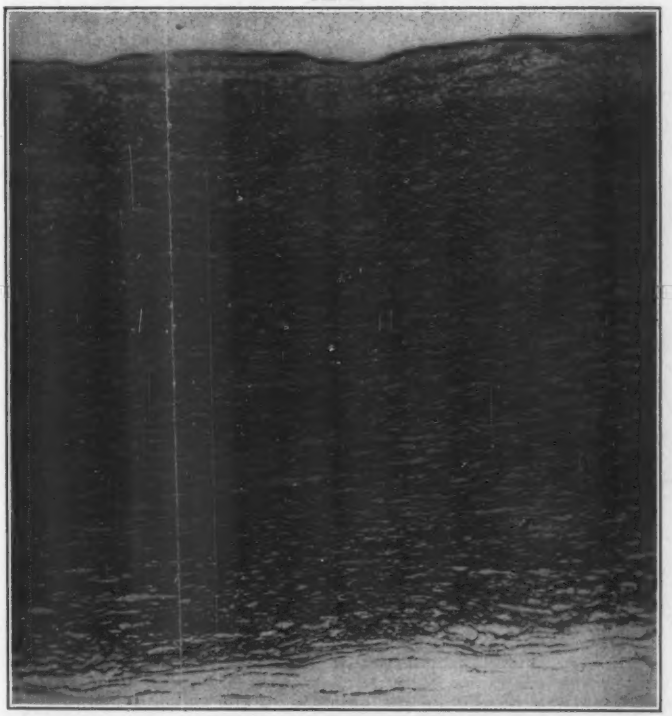
Dehydrated in

| | | |

2. Specimens treated with same strength at room temperature for twenty-four hours appeared unaffected and were put through as preceding.

3. Another set of specimens were subjected to the same strengths of the acid for seventy-two hours at room temperature and no

FIG. 6.



Tissue digestion experiments. × 185. More advanced sclerosis than preceding. Section of aorta (human) treated with 5 per cent. acetic acid. Stained with elastic-tissue selective stain. No change in tissue noted. See text, p. 870. (From the Cornell University Photomicrographic Laboratory, N. Y.)

apparent change. Further treatment was the same as preceding. (Figs. 3, 4, 5, and 6.)

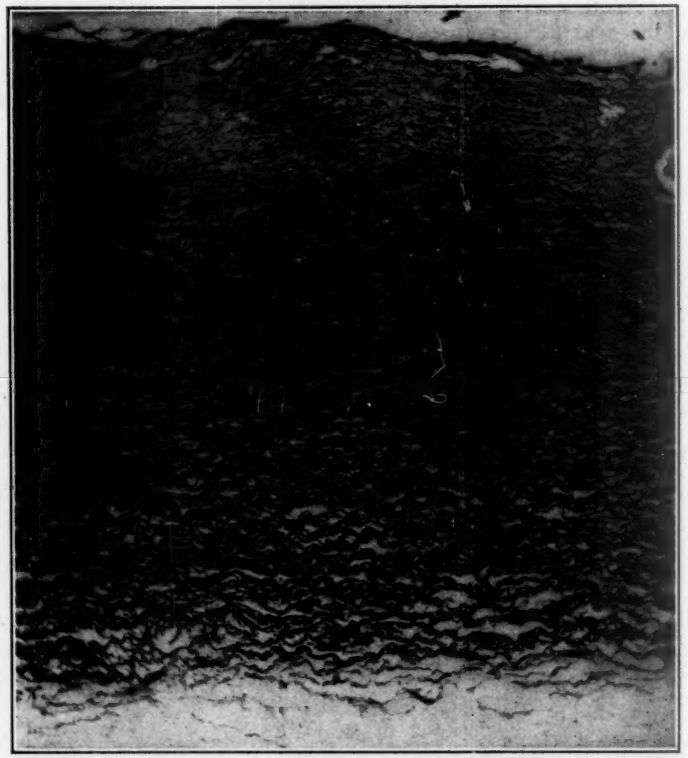
(*b*) *Potassium hydroxide*, KOH, 5, 10 and 20 per cent., in incubator for twenty-four hours, dissolved the tissue.

At room temperature for twenty-four hours, 10 and 20 per cent. partially disintegrated; 5 per cent. was left soft but intact; latter washed in running water for two hours.

Tissues were disintegrated. The strengths 5 per cent. and above are, therefore, not practical. (Figs. 7 and 8.)

Fresh material with marked arteriosclerosis was selected for another trial.

FIG. 7.



Tissue digestion experiments. × 185. Section of aorta (human) treated with 1 per cent. KOH. Stained with elastic-tissue selective stain. No change in tissue noted. See text, p. 871. (From the Cornell University Photomicrographic Laboratory, N. Y.)

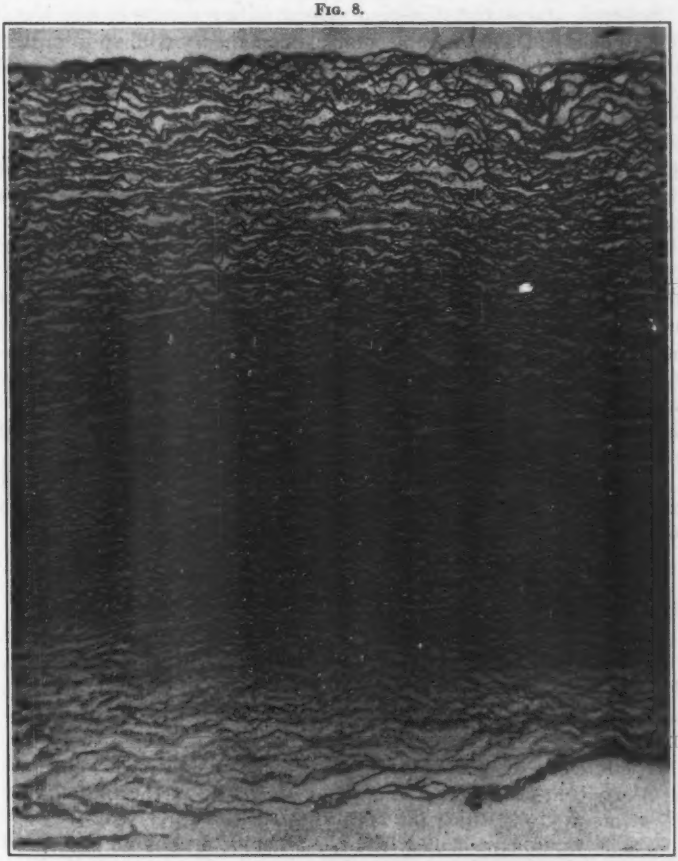
(*a*) 1. *Acetic acid*, 5, 10 and 20 per cent., in incubator twenty-four hours. Tissue softened.

| | | |

2. *Acetic acid*, 5, 10 and 20 per cent., at room temperature for twenty-four hours. Tissue quite firm and treated as above.

(*b*) *Potassium Hydroxide.* 1. KOH, 1, 2, 3, 4, 5, 10, 15 and 20 per cent. in incubator twenty-four hours; 5, 10 and 20 per cent. completely disintegrated; 4 per cent. partially broken up and fell

FIG. 8.



Tissue digestion experiments. × 185. Section of aorta (human) treated with 2 per cent. KOH. Stained with elastic-tissue selective stain. No change in tissue noted. See text, p. 871. (From the Cornell University Photomicrographic Laboratory, N. Y.)

to pieces in transferring to alcohol; 3 per cent. very soft but intact; 1 per cent. and 2 per cent. fairly firm. Last three put through same process as II. *a*.

2. KOH, 1, 2, 3, 4, 5, 10, 15 and 20 per cent. at room temperature for twenty-four hours; 10 and 20 per cent. dissolved; 5, 4

and 3 per cent., very much softened, 2 and 1 per cent. less so, but carried through the different grades of alcohol and media as preceding.

CONCLUSION. The elastic tissue having resisted the digesting agent is probably not the seat of any histological change. It has probably undergone a molecular or physical change.

Selective stains and digesting agents failing to detect any histological change leave the molecular change theory while not proven yet with points in its favor. It leaves, however, the question of vasa vasora change untouched. To this source for early nutritive changes one inevitably comes, though it cannot in all cases be shown. This question is not given such slight reference with any disregard to its importance, but the search was on other lines, and the changes in the vascular supply to the vessels is mentioned here that it may not be thought to have been overlooked.

These experiments, as far as I know, have not before been attempted. Mall's experiments in digesting, with running water, all the substances of an organ leaving only the trabeculæ are of another nature. I regret that a greater number of specimens was not subjected to these experiments, but the specimens, photomicrographs of a few of which are here presented, required many months in the preparing, as the methods are long and tedious and the material could only be of the freshest, Museum specimens being out of the question.

The work is published not as a demonstration of any important conclusion, but merely as a suggestion which may yield better results to workers in the field of physiological chemistry.

CONCLUSION.

To sum up the findings then:

1. Arterial disease appears to be rare, almost unknown in animals. Syphilis being probably peculiar to man is by this observation placed more firmly in the list of etiological factors.

2. Arterial disease in children under six years, even in those who are victims of congenital syphilis, is practically unknown. In those from six to fifteen years it is rare. It is found in the initial stage most commonly between the ages of thirty and forty years.

3. The teratologic factor, though an undeterminable one, is of great importance.

4. Arterial disease seems to be attributable to syphilis in about 32 per cent.; to tuberculosis in about 16 per cent.

5. The facts presented go to show that the colored race is affected about four times more frequently than the white.

6. General arteriosclerosis seems to be not commonly found with aneurysm and its presence may be considered as evidence against the probable development of aneurysm.

7. Staining with selective stains and treating with a chemical which digests tissue shows the elastic tissue to be free of histological alterations, suggesting that this tissue undergoes physical or molecular rather than histological change.

To Dr. Jagle, who assisted me in the histological and digesting work, I want to express my appreciation of the patience and thoroughness displayed throughout, but especially at times discouraging to us both. Also to Dr. McClellan and Professor Ewing I am indebted for valuable suggestions. From Dr. Elser I obtained practical aid in the digestion experiments, and Dr. George Warren rendered me assistance in examination of autopsy records, without which much of the material here presented could not have been collected. To Dr. Schoenberg I am indebted for aid in collecting the foreign literature.

I wish to express my appreciation of the opportunity given me by the committee of the Mütter Museum to present this report, trusting that the observations may prove of value to the practitioner and offer suggestions to those who have the opportunity of the laboratory in which to work out these problems.

REPORT OF A CASE OF LAMINECTOMY FOR GUNSHOT WOUND OF THE SPINE.

By James P. Marsh, M.D.,
SURGEON TO THE SAMARITAN HOSPITAL AND THE TROY ORPHAN ASYLUM, TROY, N. Y.

W. M. entered my service at the Samaritan Hospital August 11, 1903. He is a farmer by occupation, twenty-five years of age, white, born in the United States, and married.

He complains of severe pain at the lower part of the thorax, which extends around the body, and has some of the characteristics of a "girdle sensation." He also complains of nearly complete paralysis of his legs, especially of the left one.

The patient's father is alive and well, but his mother died of consumption. He has one sister who is alive and well, and one sister who died of la grippe.

W. M. says that he has never been ill or met with any accident excepting the one in question.

On June 14, 1903, he received a shot from a revolver just below and internal to the angle of the left scapula. The calibre of the bullet was 32. He immediately fell to the ground and could not move his legs at all for two weeks thereafter, and the bowel and bladder were also paralyzed. The wound bled but little and he at once experienced severe pain around the lower part of the thorax,

This pain has continued more or less until the present time. At the end of two weeks he began gradually to recover the use of his legs, the right one gaining more rapidly than the left. This improvement has continued up to the present, so that he can now stand erect and can move around a little by shoving one foot at a time across the floor.

This patient is well nourished and has a good muscular development. He has a recent scar just below and internal to the angle of the left scapula. Examination of the lungs, throat, heart, arteries, liver, and spleen is normal. Examination of the abdomen, kidneys, genital organs, mental condition, and cranial veins is negative. Both legs are diminished in muscular power, and the left one seems to be stiff, so that the patient does not have as good use of it as he does of the right one.

Diagnosis. Probable gunshot wound of the spine. On August 18th I took a radiograph of the spine. This showed a bullet lodged apparently in the spine on a level with the body of the eighth dorsal vertebra and a little to the left of the spinal canal.

On August 20th, he was examined by Dr. H. J. White and myself and the following notes were made: "The left pupil is larger than the right one, but both react to light and in accommodation. The tongue protrudes straight. There is no anæsthesia upon the face or neck. No jaw-jerk is present. Sensation to touch and pain upon the arms and upper portions of the thorax is normal; on the legs sensation to touch and pain is somewhat delayed, especially upon the right one. Thermic sensation is normal excepting upon the right leg, where it is somewhat confused. Analgesia is present upon the outer side of the right leg. Distinct ankle clonus is present at both ankles, and the tendon jerks are increased on both sides. There is weakness of the flexor muscles of both legs, but most marked on the left side. There is a transverse area of slight anæsthesia at the level of the twelfth dorsal and first lumbar vertebræ. A slight Babinski exists and there is patellar clonus on both sides, most marked on the right side. The plantar reflex is absent; a marked diminution in the power of the left leg is noticed and the Achilles jerk is present on both sides. In walking the feet are shoved along the floor, first one and then the other, just a little distance at a time, the left limb being very spastic and jerky."

It seemed to us that the cord had been and possibly was still pressed upon by either the bullet or a displaced portion of bone, and that an operation was indicated. Accordingly, I asked Drs. E. D. Ferguson and H. C. Gordinier to see the case in consultation with me. On August 21 Dr. Gordinier gave the patient a very careful examination and has kindly furnished me with a copy of his notes made at that time, and which are as follows: "The patient is well built and of fine musculature. There is no atrophy present. There is a rhythmic clonus of the anterior muscles of the thighs,

especially on the left side, where it involves also the calf muscles. When the patient is recumbent the feet are overextended. The patient can move the toes of each foot well, but those of the right foot more. All movements of the left leg are weak and abduction is limited, while the movements of the right leg are normal. The left leg is in slight tonic spasm. The patient cannot overcome the ordinary pressure of the hand in extending the left leg upon the thigh. The right leg equals normal. There is ataxia of the left leg; the right one equals normal; and there is no loss of muscular tone in the feet. A marked extensor spasm of the left leg occurs at intervals. Ankle clonus exists on both sides; the patellar tendon reflex is increased on both sides, but more so on the left side. There is marked patellar clonus on both sides, the left being most marked. Babinski's reflex is found on both sides, but more marked on the left. The Achilles tendon reflex is greatly exaggerated on both sides. There is slight tactile anæsthesia, to cotton, over the plantar surface of the right foot, with a strip of incomplete analgesia from the ankle along outer surface of the leg to the head of the fibula. There is no analgesia on the left leg. The temperature sense throughout the body is normal excepting for a strip on the outer side of the right leg and thigh. The umbilical reflex is absent, and there is no epigastric reflex. There are no anæsthetic areas over the anterior surface of the body. He is unable to differentiate hot and cold on the ventral surface of the right leg, ice-cold test-tubes being called warm. The upper part of the body is normal."

The eyes were examined by Dr. F. A. Smith, and he pronounced them to be normal. The urine was found to be normal.

On August 29th I made a dorsal median incision extending from the fourth dorsal to the first lumbar vertebral spines, and removed the spines and posterior arches of the seventh, eighth, and ninth dorsal vertebræ. The dura of the cord was incised for about two-and-one-half inches, and the cord appeared to be normal. Where the posterior arch of the eighth dorsal vertebra joins the transverse process on the left side was found the track of the bullet. It had not entered the spinal canal but in its passage it had forced the bone inward, so that at this point pressure had been made against the membranes of the cord. This point of protrusion into the lumen of the canal was removed. The bullet had evidently passed forward into the body of the vertebra and it did not seem wise to make further search for it. The dura was carefully sutured with tendon, the muscles were closed in layers over a small gauze drain, and the wound in the skin was closed with silk.

The pulse before anæsthesia was 90, and afterward 108. The time of anæsthesia was two hours and five minutes, the anæsthetic being at first A. C. E. mixture, followed by ether. The time of operation was one hour and fifty minutes. After the operation his temperature went as high as 101°, and his pulse varied from 130

to 60, the low point being reached on the eleventh day. The wound healed by first intention throughout, although for several days there was considerable oozing of cerebrospinal fluid from the point where the drain was removed.

On September 13th the following note was made: "Tactile sensation is greatly improved, the ankle clonus diminished, Babinski slight, ataxia in limbs improved, thermic sensations still somewhat perverted, and the muscular power in the lower limbs is stronger." On September 15th the patient was allowed to sit up in a chair and was permitted to take a few steps.

The following note was made on September 17th: "Tactile sensation is normal in the lower left limb. Just above the right ankle he is able to distinguish the dull from the sharp point. Babinski slight, lower limbs slightly ataxic, and muscular power improved. The patient can walk much better and can go up stairs much more easily than before the operation. Thermic sensation still perverted. Normal reaction to the faradic current and negative with the galvanic current."

About September 7th, the wound was found to be completely healed, but there was a very large soft swelling beneath it, which was thought to be due to cerebrospinal fluid collected outside of the membranes. This swelling became very marked and the patient complained of a great deal of headache. Consequently he was ordered to bed with high shock blocks under the foot thereof. On October 8th I aspirated from this swelling three ounces of clear cerebrospinal fluid, but the next morning the swelling was as large as ever. The condition remained about the same up to October 21st, when, as he had become very tired of hospital life, I let him return to his home in the country.

On December 29, 1903, I received a letter from him in which he says among other things: "In regard to the improvement of my case, will say that the gathering (*sic*) in my back has disappeared altogether. It disappeared within a very short while after my return home and has never returned. My headaches have also disappeared. I had several in the first week of my return, but with the disappearing of the fluid in the back my headaches went also. I am slowly but surely gaining. I go all around without a cane, but the walk is very awkward, something like one under the influence of liquor, yet I can see great improvement in that also. If I were in the dark I would not take a step or I would fall down, but now I can get around a great deal better in the dark.

"One feature of the case is very peculiar; it is the changes in my legs. The one that used to be my stronger before the operation is now my weaker one. The one I used to walk on altogether at first would break in the knee and I would fall down and the leg (the left one) feels very strong. I had to depend on it almost altogether

for a while after the operation. There is also a peculiar tightness in the left knee.

"I do all the chores here—five horses and six cows to attend. I also chop down trees, saw them up alone, and split it up into cord wood. I put up almost a cord in a day. Work does not bother my back at all, but does bother my kidneys some."

Since writing the above the patient has returned to the city and is now walking freely and without discomfiture. He is the janitor of a club-house, and is daily doing hard labor.

On March 26, 1904, I made an examination of him at my office, with the following result: The wound is firmly healed and the motions of the spine are freely and easily executed. The patient says that he has no complaints of any kind, that he feels as well as he ever did excepting for a severe cold which he now has. There is no Romberg symptom. The left great toe has a tendency to catch on the floor in the bare-foot gait, but still the walking is about perfect. The patient can place the weight of the body on either limb and swing the other leg backward and forward freely and easily. The knee-jerks are exaggerated on both sides, but the muscular power in both legs is normal. There is a slight tendency to patellar clonus on both sides and a moderate degree of ankle clonus on both sides, but it soon ceases. The cremasteric reflex is present on the right side, but absent on the left side. Both plantar reflexes are present and there is no Babinski on either side. Sensation to touch and pain in the left leg is normal, but on the external surface of the right leg to a point midway between the external malleolus and the head of the fibula sensation to pain is somewhat blunted. There is no inco-ordination in either leg; the patient can touch either patellar with the heel of the opposite foot quickly and accurately. The umbilical reflex on the left side is absent, but is present on the right side. The temperature sense is slightly confused on the right side of the abdomen and along the outside of the right leg.

Leg measurements:

Right leg	5 inches below the lower margin of patella	13 inches.
"	" at lower margin of patella	13¼ "
"	" 10 inches above the lower margin of patella	19 "
Left	" 5 inches below " "	12¾ "
"	" at lower margin of patella	13 "
"	" 10 inches above the lower margin of patella	19 "

REMARKS. In the first place, it is to be noticed that when the patient was shot he fell in his tracks and remained completely paralyzed in his legs, and also as regards his bowels and bladder, for two weeks. This should make us cautious in accepting the dictum that when this state of affairs occurs we can never expect anything like a complete recovery.

Secondly, in the performance of the operation I cannot speak too highly of the method of boldly and quickly cutting down on one side of the spines, disregarding the free hemorrhage which is sure to occur, and then, having packed the wound with gauze wrung out in very warm water, going to the other side of the spines and doing the same thing; in this way alternating from side to side until the spines and arches have been completely cleaned down to the transverse processes. This procedure reduces the loss of blood to a minimum.

Thirdly, it is to be noted that whereas the external parts of the wound healed beautifully, it is quite evident that the membranes over the cord did not unite for a considerable time thereafter. I was advised, by what I consider to be the very best of surgical authority, to reopen the wound and resuture the membranes, but subsequent events proved that it was best to have waited.

In the fourth place, I was fortunate in having a competent neurologist associated with me. Nevertheless the present neurological localization brought the lesion somewhere between the fifth and twelfth dorsal vertebræ. Indeed, then, how much of credit must be given to the Roentgen method of exact localization to the horizontal plane of the body of the eighth dorsal vertebra. Had I been equipped with Dr. Sweet's admirable localizer I undoubtedly could have located the exact position of the bullet.

GONORRHŒAL URETHRITIS, WITH UNUSUAL COMPLICATIONS.[1]

By Daniel Crosby, M.D.,
VISITING PHYSICIAN TO ALAMEDA COUNTY HOSPITAL.

Patient, a Mexican laborer, aged thirty-one years, unmarried. Admitted to Alameda County Hospital June 12, 1903; brought in by constable.

History. Little history obtainable, first, because of the somewhat delirious condition of the patient; second, because of the inability of the patient to understand English. Gonorrhœa contracted about three weeks ago. Inflammation of the eyes began about five days ago, and during these five days he lay unattended in an outhouse.

Examination of Patient. Half delirious, well-developed and well-nourished man of apparently thirty-five years. Eyelids greatly

[1] Read before the Alameda County Medical Association.

puffed, reddened, and discharging a stream of greenish-yellow pus. Conjunctivæ swollen so much as to become folded out between the lids and having the appearance of cotton placed between the lids to keep them separated. Within the lids conjunctivæ much injected and chemosis marked. Each cornea bordered by swollen conjunctiva formed a basin, as it were, which was full of pus, and the first conclusion was that the cornea was very much ulcerated. Upon cleansing the eyes each cornea was found only slightly ulcerated, but both were found very hazy.

Nose and *lips* very much swollen and congested. Thick, greenish-yellow pus discharging from nose and mouth. When this was washed away small ulcerations were to be seen not only within the nose, but also within the buccal cavity, upon the tongue, and upon the vermilion border of the lips. Salivation present. Jaws opened with great difficulty and pain. Gums spongy and retracted from all the teeth. Greenish-yellow pus exuding from every socket. Palate, pillars of fauces, and tonsils also ulcerated.

Chest shows dulness, with increased fremitus in the upper portions of both lungs anteriorly and posteriorly. Breath sounds were tubal in character. In both axillary regions dulness, decreased fremitus, and area of dulness changing boundary lines corresponding to changes in position by patient.

Respirations 42, labored and apparently painful.

Heart. No abnormality of size or position; sounds clear, accentuated. Pulse 120, full, strong.

Abdomen. Nothing abnormal.

Penis. Usual signs of acute urethritis.

Microscopic Examination. All secretions examined and all found to abound in gonococci.

Diagnosis. Acute gonorrhœa, gonorrhœal ophthalmia, and gonorrhœal infection of the nasal and buccal mucous membranes. Lobar pneumonia and pleurisy, with effusion.

In the forty-eight hours which elapsed from the entry of the patient until his dissolution the œdema and inflammation of the lids subsided somewhat, and it became easier to open them for the purpose of irrigation, even though the restless delirium of the patient prevented a very efficient course of nursing.

Autopsy. Strong, muscular individual; well nourished. Face swollen and discolored. Region of parotids and the entire scalp œdematous. Eyes, nostrils, and mouth full of pus. Eyes flattened and apparently collapsed. Nose greatly swollen and ulcerated without and within. Lips puffed up. Vermilion borders ulcerated and covered with greenish-yellow pus. Teeth can be lifted from sockets and roots are bathed with pus.

Abdomen distended; end of penis half covered with drying pus.

Intestines, stomach, and liver normal; spleen enlarged about one-third and softened; both kidneys congested and both pelves are filled with greenish-yellow pus. Ureters and bladder contain pus of same character.

The chest surface of pleuræ is coated with thick, greenish-yellow pus; left pleural cavity contains about 1000 c.c. of thin, purulent fluid; right contains about 400 c.c. of similar fluid. Large, suppurating areas occupy the greater part of the left lung and about one-half of the right; trachea and bronchi are filled with thick, creamy, greenish pus, of entirely the same character as that which has been discharging at the nose and mouth.

Pericardium contains about 75 c.c. of serum slightly tinged with blood. Heart muscle softened, but although the endocardium has lost its transparent sheen there are no indications of any ulcerations. No vegetations.

Cranial membranes under frontal lobes injected; optic thalamus markedly so. Remainder of contents of cranium not abnormal. Orbital fossa opened from behind and marked inflammatory condition noted. Ethmoidal cells are filled with pus having all the characteristics of all of the pus previously noted.

Smears from and sections of all tissues examined removed for microscopic examination. No culture made because of absence of proper culture media.

Microscopic Examination. Smears from penis, eyes, nose, mouth, ethmoidal cells, trachea, pleuræ, pelves of kidneys, and bladder without exception show diplococci which respond to Gram's decolorizing test and which have all of the characteristics, apparently, of the diplococcus of Neisser. However, some staphylococci were also present. In the secretion from the mouth a straight bacillus somewhat larger than the bacillus of tuberculosis, but not responding to the tubercle bacillus stain, was found. Aside from the straight bacilli mentioned and the staphylococci practically no bacteria were seen but the gonococci.

Sections of the suppurating areas in the lungs show that the condition of the lung is not so much a pneumonic condition as the formation of abscesses with the staphylococcus as the exciting agent.

Sections were made by Professor Ophuls, of Cooper Medical College.

I am aware of the very material defect in this report in that a culture was not made of the pus. Unfortunately, its reaction to stain does not absolutely identify this mischievous little organism. We need to see it grow. However, its staining characteristics together with the history of the case warrants us, I think, in assuming that our conclusions are correct.

In looking over the literature on the subject I do not find very

much bearing upon it. After the identification of the gonococcus by Neisser in 1879, Bumm showed that decolorizing by Gram's method is an essential procedure for the proper recognition of the diplococcus. We see references to cases of extensive infections in which the gonococcus is accompanied by many pus cocci, and one case is reported in which a patient suffered from gonorrhœa with double orchitis and general septic infection with endocarditis, in which the gonorrhœa had no part in the sepsis, but which was caused by a large dumbbell-shaped coccus which stained beautifully by Gram's method.[1]

Cardile[2] reports a pleurisy developing one month after infection with gonorrhœa. Pus, both morphologically and upon cultivation, showed characteristics of the gonococcus. Complete recovery.

West[3] refers to a *résumé* of the subject by Faitout and upon the demonstration of the gonococcus both by microscope and by culture.

Bertrand[4] says that pleurisy following gonorrhœa may be dry or effusive, usually mild and short.

Anders[5] refers to gonorrhœal arthritis with pleurisy and embolic septic pneumonia.

Lartigau[6] reports a case of gonorrhœal endocarditis in which the specific organism was demonstrated in the heart blood and in the vegetations on the valves. He refers to Thayer and Lazear, who obtained a pure culture from blood and from heart and pericardium post-mortem. Also to Rendu and Halle, who obtained a pure culture from the endometrium, from the exudate in the elbow joint, and a pure culture from the heart lesion.

Dauber and Borst[7] report malignant endocarditis of aortic valves a few weeks after the acquisition of gonorrhœa. Microscopic examination of valves, blood, abscess on aortic valve, and splenic pulp revealed abundant presence of a diplococcus which, morphologically, had all of the characteristics of a gonococcus. All nutrient media save a serum-agar tube remained sterile, but the growth upon this was not considered to be gonococci.

Lenhartz[8] obtained a pure culture from the aortic valve and with it inoculated successively a healthy human urethra.

Thompson[9] quotes Hernig as having demonstrated the diplococcus upon the heart valves, and Councilman as having found them in the cardiac muscle.

Jicinsky[10] reports a case of pleurisy following gonorrhœa, but his conclusions are not substantiated by cultures.

Jessionek[11] reports a case of stomatitis preceded by double gonorrhœal conjunctivitis. He describes round, grayish-white patches on the surface of the tongue and cheeks. Much swelling and soreness and evidence of inflammation of sublingual glands. Cover-glass preparations and cultures showed gonococcus.

Sidney Vines[12] reports a case of gonorrhœal gingivitis following

gonorrhœal urethritis: "Infection of urethra March 22d. April 16th, complained of sore mouth and having to spit often. Gums red, swollen, spongy; an overabundant supply of saliva. Condition resembled a mercurial stomatitis. By April 20th he presented an appalling spectacle. Huge, incapable, with big head, of bull-dog sort, the lower jaw drooping, saliva unceasingly trickling from the angles of his mouth, and every tooth loose and bathed in pus which oozed from every socket. He could not eat, and drank with difficulty. Temperature, 101°. Atmosphere of the bedside very offensive. April 22d, gonococci demonstrated in the pus. May 10th, mouth normal." This patient had been in the habit of picking his teeth, every day after eating, with a splinter of wood which he had whittled down.

Holder [13] reports the case of a man who had buccal coitus with a man suffering with gonorrhœa. On the next day he complained of pain in the lips and gums. On the fourth day mucous membrane of lips and buccal cavity intensely red; gums spongy, with a tendency to bleeding and receding from the teeth, and the buccal secretion became much increased in quantity. Motion of the mouth was very painful. Holder states that the affection begins with a sensation of heat and dryness of the mouth, which at first appears very red. Soon a purulent discharge flows from the swollen and inflamed parts, which may be covered with an aphthous-like exudation.

Cutler[14] reports a case of gonorrhœa of the mouth following coitus ab ore. On morning following, mouth raw and sore and with "horrible" taste. On second day little sores appeared on lips, and on third day the gums and tongue were swollen and painful. By the fifth day the whole buccal cavity was so inflamed that she could not eat and a whitish fluid mixed with blood and having a very unpleasant odor was secreted.

Examination. Mucous membrane of lips and cheeks red, denuded of epithelium in spots and covered with specks of false membrane which were readily detached, leaving an excoriated surface. Gums swollen, retracted from teeth, and bled very readily on pressure. Tongue swollen and very tender. It could be only slightly protruded and then only with much effort and pain. Its surface was red and glazed and covered with small ulcers which secreted a thick, yellow pus. The soft palate and pillars of the fauces were very much inflamed, but parts beyond were in normal condition. Breath was very offensive. Little salivation. The mouth secretion was seen to consist of pus and epithelial cells and a large quantity of bacteria. In the false membrane an organism resembling the gonococcus was found, but its identity was not established absolutely.

So much for the recital of reports of infection by the gonococcus,

It is of some interest to note the extension of the disease in the patient under consideration.

The urethral infection was undoubtedly the primary one and that of the eyes next. It is logical to suppose that the infection went thence to the nose and mouth. The infection of the lung was apparently not gonorrhœic in character, although the pus in the pleura had entirely the microscopic appearance of gonorrhœal pus. This may also be said of pus that was removed from the trachea above its bifurcation.

There was no evidence of endocarditis; so the trouble in the lung can perhaps be referred less to the blood stream than to inspiration.

With reference to the portals of infection of the pleura Grober records some experiments that are of some interest. He found that forcible inhalation of finely powdered stains resulted in the appearance of granules in the parietal and visceral pleura and the discoloration of the lymph channels along the borders of the ribs. The same experimenter injected stains into tonsils and found stain in lymph channels and glands down the neck and in lymph channels of the lower mediastinum.

This may, perhaps, give us a clue to the origin of the gonococcus infection of the pleura; but whether from this path or the blood stream we are forced only to conjecture.

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REVIEWS.

A Compend of the Practice of Medicine. By Daniel E. Hughes, M.D., Late Chief Resident Physician, Philadelphia Hospital; Late Physician-in-Chief, Insane Department, Philadelphia Hospital; Formerly Demonstrator of Clinical Medicine in the Jefferson Medical College of Philadelphia, etc. Seventh Revised Edition, Edited, Revised and in Parts Rewritten by Samuel Horton Brown, M.D., Assistant Dermatologist, Philadelphia Hospital; Assistant Dermatologist, University Hospital Dispensary, etc. Including a Section on Mental Diseases and a very Complete Section on Skin Diseases. Illustrated. Philadelphia: P. Blakiston's Son & Co., 1904.

This posthumous edition of Dr. Hughes' book has been considerably rewritten and rearranged, as stated by the editor in his preface. It has thus been modernized and contains the latest theories and views on each subject. It needs but a brief mention of Dr. Hughes' exceptional opportunities for the study of disease in all its manifestations to show how peculiarly fitted he was to produce a work on general medicine. Dr. Hughes was for many years Chief Resident Physician at the Philadelphia Hospital, at which place he saw and had under constant observation not only numerous cases of the acute diseases, but an unusually varied and large assortment of chronic diseases, particularly nervous diseases. He also had opportunities to study a great number of cases of mental diseases of all varieties and in all stages. He was also able to widen his views through contact with the attending physicians, who in many cases had other sources for gaining experience.

So many topics are considered in this compend that a bulky volume necessarily results, but the use of thin paper and flexible covers make it surprisingly compact and easy to read. The consideration of each subject is divided into paragraphs devoted to the etiology, pathology, prognosis, differential diagnosis, and treatment; and while unessential matters are only briefly mentioned, and in no case is there a long description of any one feature, an excellent account of each disease is given. The characters that make the book of most use from a practical point of view are the numerous prescriptions and suggestions for treatment and the

paragraphs on differential diagnosis which mention and contrast the various conditions which may be confounded. The subjects are grouped under the proper divisions and a brief general discussion precedes each section, while in the case of gastrointestinal and renal diseases and blood diseases the diagnostic technique is described so that methods of clinical diagnosis in common use are to be found.

The volume is so arranged and its contents are of such a high character that it should be of service to the physician whose time is so occupied that he cannot consult the larger text-books, or to one who wishes merely to refresh his knowledge and to fix in his mind the essential facts freed from details. F. W. S.

BURDETT'S HOSPITALS AND CHARITIES, 1905: BEING THE YEAR BOOK OF PHILANTHROPY AND THE HOSPITAL ANNUAL. By SIR HENRY BURDETT, K.C.B. London: The Scientific Press, (Ltd.).

THIS invaluable work has established itself so firmly as an absolute necessity for those who are interested in hospitals and charitable enterprises that it hardly needs an extended review. It not only presents a street directory, but it gives the executive staffs, the number of patients, income and expenditure of practically every hospital and institution dealt with. Each year has seen some improvement in the general arrangement of the work, rendering it more useful, and the vast amount of information within it more accessible. It covers practically every charitable institution in Great Britain, the British Colonies, and the United States, including not only hospitals and institutions concerned in the care of the sick, but also orphanages, homes, and aid and rescue societies. The information contained within it is so carefully compiled from authoritative sources that it may be absolutely relied on. J. H. G.

RADIOTHERAPY AND PHOTOTHERAPY. By CHARLES WARRENNE ALLEN, M.D. Illustrated with 131 engravings and 27 plates in colors and monochrome. New York and Philadelphia: Lea Brothers & Co., 1904.

DR. ALLEN has devoted the major part of his work to radiotherapy, in which his experience with the Roentgen rays is most extensive. He gives sufficient explanation of the principles and apparatus to render the subject clear to his readers without too much elabora-

tion of detail, which is often more confusing than helpful. His chapter on methods of administration and protection are valuable to every operator, while his warning against the dangers to the operator himself are timely, though possibly not sufficiently strong. This subject is one which should receive special emphasis from every author who desires to further the best interests of those he would instruct. Medical and surgical diagnosis receive a concise review as is fitting in a work primarily devoted to therapy.

In the description of technique, the author appreciates keenly the necessity of developing a more accurate method of dosage, and strives to indicate methods by which it can be attained. Progress is the characteristic of this method of therapy at the present time, and so rapid is this progress that it demands almost quarterly editions to keep pace with the advances in technique. More accurate instruments for measuring dose are already at hand, and their character and use will doubtless appear in the next edition, as also the variations in technique, which adapt this therapeutic agent to the various manifestations of malignant disease in superficial and deep situations. The reason why therapeutic action takes place on the opposite side of the surface treated, as the ear or nose, will then be explained.

The chapters on Light, Actinotherapy, Radioactivity, and High-frequency Currents are timely, but serve to show that those therapeutic agents are inferior to the Roentgen rays, both because they are much slower in action and because their potency is vastly inferior.

The work, on the whole, leaves little to be desired, though it is fair to say that at the present time the best views and latest technique can hardly be expected outside of current literature. It is impossible to teach experience in a book, but the author's aim should be to give his readers all the knowledge he can gather that will further their search for experience. C. L. L.

MANUAL OF PHYSIOLOGICAL AND CLINICAL CHEMISTRY. By ELIAS H. BARTLEY, B.S., M.D., Ph.G., Professor of Chemistry, Toxicology and Pediatrics in the Long Island College Hospital; author of *Medical and Pharmaceutical Chemistry*. Second edition, revised and enlarged, with 47 illustrations. Philadelphia: P. Blakiston's Son & Co., 1904.

THIS book of 188 pages contains a great many things and many more subjects are treated than one would judge from its title. A large part of the book is taken up by a description of methods used in clinical chemistry and clinical diagnosis. Many of the methods

are given incompletely, or in such a way as to be valueless as a laboratory guide. At times comments are made upon the clinical significance of the various findings by the methods, but these additions seem to be at random. A number of laboratory experiments to bring out the properties of various organic substances are found from time to time as one reads the book. Clinical examination of the blood is treated in five and one-half pages, and although the author apologizes for the section in the preface it is obvious that he would have done better to have omitted it. Other subjects of slight importance or of questionable clinical application are treated at some length. For instance, several pages are devoted to cryoscopy of the urine. Milk modification is also discussed.

The book is without a table of contents, and its arrangement is poor. It is difficult to see to what class of readers the book would prove of use. G. C. R.

THE TREATMENT OF FRACTURES: WITH NOTES UPON A FEW COMMON DISLOCATIONS. By CHARLES LOCKE SCUDDER, M.D., Surgeon to the Massachusetts General Hospital. Fourth edition, thoroughly revised, with 688 illustrations. Philadelphia, New York, and London: W. B. Saunders & Co.

THE fact that this book has passed through four editions since it first appeared in 1900 is sufficient proof of its worth. In addition to some alterations in the text, there has been considerable change and improvement made in the illustrations. An entirely new chapter, but a brief one, on the subject of Some of the Common Dislocations, has been added. This book certainly presents the subject of the treatment of fractures in a most thorough and complete manner. It is to be especially commended upon the clearness of the text and the excellence of the illustrations, which enable the reader to understand and practice the various manipulations and dressings described.

We feel that we must repeat a criticism made of one of the earlier editions, and that is that we think the time suggested for active and passive movements of the fingers and wrist in fracture about the lower end of the radius is not early enough. We believe that the movement of the fingers in these fractures should be commenced practically at the first dressing, since, if properly done, there is not the slightest danger of displacement of the fragment, and the restoration of function is much more prompt. Another criticism that is of minor importance, but which might be mentioned, is that the author speaks of the angular splint applied to the front of the upper extremity as an "internal" angular instead of "anterior" angular.

Taking it as a whole, this is one of the best works on the treatment of fractures with which we are acquainted. J. H. G.

GALLSTONES AND THEIR SURGICAL TREATMENT. By B. G. A. MOYNIHAN, M.S. (London), F.R.C.S. Philadelphia, New York, and London: W. B. Saunders & Co., 1905.

THE author of this volume need make no apology for devoting so much space to the single subject of gallstones, and the reader will certainly not think of asking for one. The subject is most important both from the point of view of the surgeon and that of the internist, and therefore a practical dissertation such as this is of the greatest value. The author has had an unusually large experience in the surgery of the gall-bladder and its associated organs, and is consequently thoroughly fit for the task. The book should be read by medical men in order that they may see the results of neglected gallstone disease and compare the operative treatment of such cases with that of the uncomplicated ones, and also in order that they may understand the symptoms to which the surgeon attaches most value in the catastrophies which occasionally take place in diseases of the gall-bladder.

The surgeon can learn much from a careful study of Moynihan's technique, which is described very fully and comprehensively. After discussing the anatomy of the gall-bladder and ducts and the varieties of gallstones, considerable space is devoted to the pathology and especially to the symptoms of gallstone disease.

The portion of the book dealing with this subject is beautifully illustrated, which greatly enhances the value of the work.

All of the various operations upon the gall-bladder are described in detail. The reader will observe that the author, like so many surgeons of extensive experience in this line of work, has greatly extended the indications for the operation of cholecystectomy.

The work we can heartily recommend as most satisfactory and of the greatest practical value, especially to the surgeon.

J. H. G.

A TREATISE ON DISEASES OF THE NERVOUS SYSTEM. By L. HARRISON METTLER, A.M., M.D. Chicago: Cleveland Press, 1905.

DR. METTLER believes that "the neurone doctrine is now an accepted fact," and that "its teachings have done more to illuminate the dark places of neurology than has any single scientific generalization heretofore promulgated." He bases his work on these two statements and hopes that the student will be able "to behold the entire field of neurology under the brilliant illumination cast upon it by this scientific generalization." It is a dangerous thing to base a medical book upon a theory, because theories have a habit of going out of fashion and the neurone doctrine is not as

fashionable as it was a few years ago. Apart altogether from theory the descriptions of disease are for the most part clear and the therapeutic directions excellent. We hope that when a new edition is issued lighter paper will be used, because it is cruel to expect anyone to hold and study a book weighing six and a half pounds.

C. W. B.

ELECTRICITY IN EVERY-DAY LIFE. By EDWIN J. HOUSTON, Ph.D. In three volumes; illustrated. New York: P. F. Collier & Son.

ALTHOUGH these three volumes are hardly to be considered as medical works, they nevertheless contain information which is of value to every physician who is desirous of availing himself of the most recent knowledge in the ever-progressing subject of electricity. The applications of electricity to medical science are increasing daily. It is impossible for a doctor to really study the subject, and a book such as this enables him to grasp the essentials necessary to a working knowledge and enables him to use the various electric appliances with which every well-to-do office is equipped with some understanding of the nature of the elements which he employs. Dr. Houston's explanations of the scientific problems involved and his descriptions of apparatuses and technique are so practical and clear that a layman can grasp them and apply them to his uses with the greatest ease. Aside from its technical value the book is charming reading, and its delightful accounts of the wonderful discoveries and inventions which have succeeded one another since Benjamin Franklin's famous kite-flying experiment will speak to all and stimulate a desire to know more of a wonderful subject.

F. R. P.

THE DOCTOR'S RED LAMP. A BOOK OF SHORT STORIES CONCERNING THE DOCTOR'S DAILY LIFE. Selected by CHARLES WELLS MOULTON. Chicago, Akron (Ohio), and New York: The Saalfield Publishing Co., 1904.

THIS book contains twenty-two short stories dealing with the life of the doctor. Curiously enough, the stories are almost all of them by laymen. As the editor says in his preface, many of them are old favorites, a fact which will probably appeal all the more to those who see the book. They have been well chosen, and their perusal will, we hope, give many a physician a number of pleasant hours of recreation. The idea of a series of books of this nature

is a good one and its execution is commendable. If the editors continue to show the discretion and good sense which is manifested in their choice of material, the books should commend themselves to every physician. The volume is accompanied by four handsome illustrations appropriate to the text. F. R. P.

APPLETON'S MEDICAL DICTIONARY: AN ILLUSTRATED DICTIONARY OF MEDICINE AND ALLIED SUBJECTS IN WHICH ARE GIVEN THE DERIVATIONS, ACCENTUATION, AND DEFINITION OF TERMS USED THROUGHOUT THE ENTIRE FIELD OF MEDICAL SCIENCE. Edited by FRANK P. FOSTER, M.D., editor of *The New York Medical Journal* and *Philadelphia Medical Journal*, consolidated; of a *Reference Book of Practical Therapeutics;* and of *Foster's Illustrated Encyclopædic Medical Dictionary.* New York and London: D. Appleton & Co., 1904.

BECAUSE of the rapid advance which has been made in nearly all lines of medicine and surgery during recent years there has arisen a demand for newer and more complete medical dictionaries. It has been the aim of the publishers and editor of this volume to satisfy this demand, and that they have done it there can be little doubt.

The book contains an enormous amount of information, especially regarding anatomy and diseases. It has not adopted some of the newer methods of spelling which have become so prevalent, but for this we can only commend it. The definitions are very clear and satisfactory.

The one criticism we would make is that probably too much has been put under single headings and consequently the person who consults the book for a definition instead of finding the word in large type may have to look through possibly a page of subheadings; as for instance, Littre's hernia, which is indexed under hernia, but not under Littre. Richter's hernia we do not find mentioned at all. Neither Bright's disease nor Graves' disease can be found excepting under the general heading of Disease, there being no cross-reference to the same. We believe it to be wrong to attach proper names to diseases and operations, but nevertheless certain of these have become so firmly fixed in our nomenclature that they should at least be given a cross-reference.

Notwithstanding this criticism, which of course does not depict a serious defect in the work, we believe that this dictionary will be found a most satisfactory book of reference. The illustrations are not so frequent as in most modern dictionaries, but illustrations are not nearly so essential to a dictionary as to a modern text-book.

J. H. G.

MENTAL DEFECTIVES, THEIR HISTORY, TREATMENT, AND TRAINING. By MARTIN W. BARR, M.D. Philadelphia: P. Blakiston's Son & Co., 1905.

DR. BARR has lived with and studied "mental defectives" for about twenty years; a book therefore written by him ought to be of value, and this is one of the most practical and useful books for the care of the feeble-minded that has been written. The keynote of the book is the following: the hopelessness of cure and the needless waste of energy in attempting to teach an idiot, what may be attained in training the imbecile, and the urgent need of preventing the backward child from degenerating into imbecility, and finally the necessity of safe-guarding the immoral imbecile from crime. No attempt is made to explain the pathology and morbid anatomy of idiocy and imbecility.

Dr. Barr is strongly in favor of asexualization as a means of preventing the propagation of idiots and imbeciles. He believes these conditions are largely hereditary and that the State should appoint surgeons and neurologists to operate non-improvable cases in institutions. If such a function as this properly belongs to the State, then it ought also to asexualize paranoiacs, many cases of insanity, and not a few alcoholics and hysterics, for all of these propagate degenerates. Very few idiots propagate, and the imbeciles that are sexually strongest are those of the higher grade who are or seem to be improvable. The State Assembly has just passed a law doing what Dr. Barr desires. It is now awaiting the Governor's decision. We think the law pernicious because it extends the functions of the State too much and thus no good will come from it as the dangerous cases will not come under it.

A very large part of the book is filled with the histories of illustrative cases. Every one interested in mental defectives and their care, whether physicians, judges, teachers, or persons interested in the study of juvenile crime, should read and own this book.

C. W. B.

ESSENTIALS OF NERVOUS DISEASES AND INSANITY, THEIR SYMPTOMS AND TREATMENT. By JOHN C. SHAW, M.D. Fourth edition, thoroughly revised by SMITH ELY JELLIFFE, M.D., Ph.D. Philadelphia, New York, and London: W. B. Saunders & Co., 1904.

THIS is a very good little book for the purpose of which it was intended, namely, as a primer for advanced students. It concerns itself principally with diagnosis. All anatomical and physiological details are omitted. The first 150 pages are devoted to nervous diseases, the next 40 to mental diseases. It is written in clear English and the illustrations are very good. C. W. B.

ATLAS AND EPITOME OF GENERAL PATHOLOGICAL HISTOLOGY. By DOCENT DR. HERMANN DURCK, of the Pathological Institute, Munich. Authorized translation from the German. Edited by LUDWIG HEKTOEN, M.D., Professor of Pathology in Rush Medical College, Chicago. With 176 colored illustrations and 80 lithographic plates and 36 figures in black and colors. Philadelphia, New York, and London: W. B. Saunders & Co., 1904.

To anyone who has been familiar with the exquisite colored plates in the original edition of Dürck's *Atlas and Epitome of General Pathological Histology*, the present English edition, edited by Dr. Hektoen, will be exceedingly gratifying. The beautiful illustrations, reproduced with the greatest care and accuracy, are almost sufficient in themselves to teach the subject. The accompanying text is, however, only explanatory, and reviews the vast field of general pathology in a brief and lucid manner. The original suffers few changes in the translation. Occasionally the editor has inserted short notes, which always add to the value of the paragraph.

The student of pathology, who does not read German, is fortunate to have this book made available for his use. W. T. L.

THE MEDICAL EPITOME SERIES. SURGERY. A Manual for Students and Practitioners. By M. D'ARCY MAGEE, A.M., M.D., Demonstrator of Surgery and Lecturer on Minor Surgery in the Georgetown University Medical School, and WALLACE JOHNSON, PH.D., M.D., Demonstrator of Pathology and Bacteriology, Georgetown University Medical School. With an Appendix on *x*-ray Work in Surgery by EDWARD O. PARKER, A.M., M.D., Physician at the New York Dispensary. Series edited by V. C. PEDERSEN, A.M., M.D. Illustrated with 129 engravings. Philadelphia and New York: Lea Brothers & Co.

ONE can easily realize the difficulty of epitomizing general surgery in so small a book as the present volume, and possibly anyone undertaking such a work will be criticised by his reviewers for devoting too little space to some subjects and too much to others. The present book is open to this criticism, as can be seen by a reference to the chapter on Fractures and to that on the *x*-rays.

In most respects, however, the book is very satisfactory. The illustrations are especially good. We believe that the list of questions at the end of each chapter is a good idea, as it causes the student to review the text and also to formulate some idea of the subjects which he has studied. The views expressed by the author are in accord with the most advanced surgical authorities, and are expressed in a way well adapted to the need of the student. J. H. G.

ESSENTIALS OF MATERIA MEDICA, THERAPEUTICS, AND PRESCRIPTION WRITING. By HENRY MORRIS, M.D., Fellow of the College of Physicians of Philadelphia, etc. Sixth edition, thoroughly revised by W. A. BASTEDO, PH.G., M.D., Tutor in Materia Medica and Pharmacology at Columbia University (College of Physicians and Surgeons), New York, etc. Philadelphia, New York, and London: W. B. Saunders & Co., 1904.

THIS little book bears evidence of careful revising and should be useful for a review of the subjects treated therein. We imagine, also, that examiners may find it convenient in framing papers. While it can never supplant the systematic text-book, for it lacks breadth and perspective, the care in compilation and conciseness of expression have made for it a field of usefulness of which its popularity is good evidence. R. W. W.

MULTIPLE PERSONALITY, AN EXPERIMENTAL INVESTIGATION INTO THE NATURE OF HUMAN INDIVIDUALITY. By BORIS SIDIS, M.A., Ph.D. (Harvard), and SIMON P. GOODHART, Ph.B. (Yale), M.D. New York: D. Appleton & Co., 1905.

THIS book is interesting but unsatisfying. One takes it up hoping to have solved one of the greatest problems of psychology and having read it is no wiser than before. One hundred and fifty-three pages are taken up with the history of one case. This is interesting reading, but one feels he is at the mercy of the intellectual accuracy of the patient, and people who pass through such experiences as he did are not always accurate in their statements. There is growing up a school of mystical materialism, if one may couple two words so apparently contradictory, and this book seems to belong to that school. It is very interesting, but "the nature of human individuality" is not explained by it. C. W. B.

BEAUTY THROUGH HYGIENE. By EMMA E. WALKER, M.D. New York: A. S. Barnes & Co., 1904.

THIS volume, one of the *Woman's Home Library* series, is a collection of clearly written articles on "Common-sense Ways to Health for Girls," as the title-page announces. To readers of a prominent magazine for women portions of the book will seem familiar, but certain truths bear repeating, and in this case Truth and Beauty are indeed one. While probably few women would have the time, and fewer still the patience to carry out all the doubt-

less valuable suggestions contained in these pages, yet most of the advice is worth taking, and ought to appeal not only to the woman reader, but also to those who have the guidance of young girls who are at the age when the formation of daily habits of personal hygiene is most important. J. P. G.

DISEASES OF THE LIVER, GALL-BLADDER, AND BILE-DUCTS. By H. D. ROLLESTON, M.A., M.D. (Cantab.), F.R.C.P., Physician to St. George's Hospital, London; Formerly Examiner in Medicine in the University of Durham; and Fellow of St. John's College, Cambridge, England. Fully illustrated. Philadelphia, New York, and London: W. B. Saunders & Co., 1905.

THERE have been many good books written on the liver—Glisson in the seventeenth, Bianchi in the eighteenth, Frerichs, Budd, and Murchison in the nineteenth centuries. Of modern works, that of Frerichs, issued in 1858 and reproduced in the New Sydenham Society's publications, has remained without rival in any language, and now this treatise of Rolleston's easily takes a place beside it. There has not been for years issued from the press so comprehensive and accurate a consideration of the diseases of an important organ. In every way it will rank as a masterpiece, and reflects the highest credit upon British medicine. Dr. Rolleston has been a well-known student of diseases of the liver, but very often the special concentration on a subject unfits a man for a wide view of its problems. But this is in every way a model work, showing an unusually large personal experience with a range of reading quite unusual, even in these days of accurate bibliography. It would be difficult to mention a recent work which represents so truly the cosmopolitan character of medicine. Tribute has been collected from every country, and had Dr. Rolleston been a Frenchman, a German, or an American he could not have shown greater familiarity with the native literatures. American contributions to the subject are most fully and satisfactorily represented. It is remarkable how familiar the author is with the writings of physicians on this side of the water.

It is difficult to select a special section for commendation where all are so good, but the concluding chapter on cholelithiasis, extending over some seventy pages, may be referred to as a model of its kind. The subject is treated with great fulness, and equal consideration given to the medical and chirurgical sides of the many problems which are at present before the profession.

The writer's style is admirable, clear, and to the point, and his judgment on questions is enforced by an evident desire for fairness. The work should appeal to a large class of readers. It will take its place on the book-shelves of every careful practitioner who wishes

a helpful guide in this important class of diseases. To the surgeon and specialist it will prove invaluable, while to the student of the many problems in hepatic pathology still awaiting solution it will prove an authentic guide of the highest order. E. Y. D.

THE SURGERY OF THE DISEASES OF THE APPENDIX VERMIFORMIS AND THEIR COMPLICATIONS. By WILLIAM HENRY BATTLE, F.R.C.S., Surgeon to St. Thomas' Hospital, etc., and ELDRED M. CORNER, M.B., B.C., F.R.C.S., Surgeon in Charge of Out Patients to St. Thomas' Hospital, etc. Chicago: W. T. Keener Co., 1905.

THIS small volume, comprising about 200 pages, contains some very useful information on the subject of appendicitis, which it states in a very clear and concise manner. A brief, interesting history of the disease is given, and considerable space is allotted to the discussion of the diagnosis and the various complications of the disease. The methods for the removal of the appendix are described, especially the one practised by the authors—*i. e.*, "temporary displacement of the rectus abdominis." The last chapter is devoted to the subject of the relation of appendicitis to the selection for life insurance, and a number of rules for insurance examiners are given. The book is well written and we can highly commend it to surgeons as well as general practitioners. C. F. M.

ELECTRICITE MEDICALE. Par le DOCTEUR H. GUILLEMINOT. Avec 79 figures dans le texte et 8 planches hors texte. Paris: G. Steinheil, 1905.

IN this book of 613 pages the author has given on the whole an excellent and conservative description of modern electrotherapeutics. In addition to those subjects, including the *x*-rays, usually treated of in such works, we find considerable attention given to the effects and employment therapeutically of agents having electricity as a generator—*i. e.*, ozone, vibratory massage, phototherapy, and thermotherapy. The newer forms of the electric current employed in medicine, as the sinusoidal, undulatory, and high-frequency, receive considerable attention. The sections upon the physics, physiological action, and therapeutics of currents of high frequency are of especial value, as would be expected from a French author, the electrotherapeutists of that nation having done so much for the advancement of this modality as a therapeutic agent. Under the description of the reaction of degeneration considerable space is devoted to possible explanations of the reasons for

the occurrence of this phenomenon and the experiments of Wiener, May, and Joteyko are fully detailed. A novel feature of the book is that instead of devoting a separate section to electrodiagnosis this is discussed in all cases in which the electricity is of aid in the diagnosis in connection with the description of the therapeutic procedures of use in the disease. This is an innovation in arrangement of considerable convenience. In this connection it is peculiar that two such valuable diagnostic aids as the myotonic and myasthenic reactions are not described. In discussing the treatment of aneurysm by electrolysis the excellent work of American operators, as D. D. Stewart, Hunner, and others, is not mentioned. The illustrations are not numerous, but among them are some very useful plates showing the motor points. These differ from those usually found in the text-books in that the motor points of all the muscles supplied by a certain nerve are represented by the same color; thus the motor points of all those muscles supplied by the median nerve are colored red, by the ulnar blue, and so on. This arrangement has also been independently adopted by Mosher, of Albany, in his work on electrodiagnosis. In conclusion it can be said that this book will prove of service to all interested in electrotherapeutics. C. S. P.

PNEUMONIA AND PNEUMOCOCCUS INFECTIONS. By ROBERT B. PREBLE, A.B., M.D., Professor of Medicine, Northwestern University. Illustrated. Chicago: Cloyd J. Head & Co., 1905.

DR. PREBLE'S subject is handled in the most satisfactory manner, the book considering pneumonia carefully in every aspect. A series of 3600 cases of pneumonia reported by six observers has been collected by the author, and these cases have been used in the compilation of statistical tables and charts bearing on almost every feature of the disease. By taking such a large number of cases reported by so small a number of men a high degree of uniformity of observation is secured; and since pneumonia differs to such a marked extent in different localities and in different years the average percentages are made nearly exact by the fact that the individual reporters are of different nationalities and report cases occurring in different epidemics. It is of distinct advantage to have definite figures showing the average frequency of occurrence of the various symptoms and complications and the average time at which these are likely to appear, and such figures are not readily found.

Outside of the statistical features there is little new or original matter in the book, but so much space is devoted to each symptom

and complication, with complete and practical descriptions of the diagnostic signs and technique in each instance, that the reader may obtain valuable information on this disease, which is undoubtedly becoming more prevalent.

A section is given to the study of the pneumococcus infections which may occur either independently or coincident with lesions in the lungs. In this connection it is urged that the blood culture and lumbar puncture be used in all doubtful cases in addition to the ordinary methods of diagnosis. Cases are cited in which pneumococcus meningitis and arthritis occurred without any pulmonary manifestations of the infection, and were mistakenly diagnosed as epidemic meningitis and rheumatism respectively, until blood cultures had been made.

In the section on treatment the various theories or modes of treating pneumonia which have been highly praised are discussed, and a statement of the practical results obtained at the hands of others than the discoverers is given. The author states as his opinion that no plan of treatment which has been advocated has so far proven to be specific in the test of frequent trials in varying places. He recommends symptomatic treatment as the method most likely to assist and least likely to injure the patient, and says that no treatment can be considered specific or thoroughly satisfactory until a reliable pneumococcus antitoxin has been perfected.

F. W. S.

ESSENTIALS OF BACTERIOLOGY, BEING A CONCISE AND SYSTEMATIC INTRODUCTION TO THE STUDY OF MICRO-ORGANISMS. By M. V. BALL, M.D. Fifth edition, thoroughly revised by KARL M. VOGEL, M.D., Assistant in Pathology, College of Physicians and Surgeons, Columbia University, New York City. Philadelphia, New York, and London: W. B. Saunders & Co., 1904.

THE author and editor have succeeded in bringing together in a systematic manner the essentials needed in the study of bacteria. The style employed is concise, much of the book being written in the form of more or less expanded definitions.

Part I. consists of general considerations and technique, the latter subject being quite fully and well treated, forming the best part of the book. In considering the various bacteria, perhaps undue space is given to the non-pathogenic varieties, of which about thirty are discussed. A special chapter is devoted to the bacteria pathogenic for animals, but not for man, and the pathogenic protozoa are described. In an appendix various technical subjects, such as the examinations of air, water and soil, milk, and of the organs and cavities of the human body, are briefly discussed, and

a tabulation of both pathogenic and non-pathogenic organisms is given. The book is 243 pages in length and covers consisely the subject of bacteriology with a considerable degree of completeness, bringing it in most particulars up to date. G. C. R.

A Manual of Experimental Physiology for Students of Medicine. By Winfield S. Hall, Ph.D., M.D. (Leipzig); Professor of Physiology, Northwestern University Medical School, etc. Philadelphia and New York: Lea Brothers & Co.

This book is the published form of a manual which the author has used for some years in his teaching. It contains a series of experiments which quite thoroughly cover the field of physiology as taught to the medical students of to-day. The first chapter is on "Cytology," and the experiments in it are devised to bring out the fundamental activities of single-cell organisms, cilia, and the effects of various substances upon simple organic life. Electric apparatus is carefully explained in the following chapter dealing with the general physiology of muscle and nerve tissue. Chapters upon the circulation and respiration, normal hæmatology, digestion and absorption, vision, the nervous and muscular systems, complete the book.

The method generally employed is to explain first the appliances, then describe the methods of preparation and technique of the operations, and, lastly, the special points to be observed are given.

The book, which contains 245 pages, is especially valuable because, unlike the usual text-book upon physiology, it is stamped with the individuality of the teacher. G. C. R.

Surgery of the Prostate, Pancreas, Diaphragm, Spleen, Thyroid, and Hydrocephalus. An Historical Review. By Benjamin Merrill Ricketts, Ph.B., M.D. Cincinnati, 1904.

An index to the surgery of the prostate, pancreas, thyroid, etc., would be a more comprehensive name for this book, as its pages are principally made up of references chronologically arranged from the literature pertaining to these different organs. A chapter is allotted to each organ; a brief description of its anatomy, etiology, and its pathological conditions are given, and the various surgical operations are described. The bibliography comprises the main part of the book and shows very careful and exhaustive work on the part of the author. C. F. M.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Heart Affections in Scarlet Fever.—SCHMOLTZ (*Münchener med. Wochenschrift*, 1904, li. p. 1417) states while the cardiac lesions in diphtheria have been carefully studied, those occurring during and after scarlet fever have been more or less overlooked, owing probably to the infrequency of such serious and tragical results as follow diphtheria. In a study of 191 cases of scarlet fever, Schmoltz, of Dresden, found that about 35 per cent. showed signs of some circulatory disturbance. In the beginning of the attack the pulse is always rapid. This does not indicate any cardiac derangement. Indeed, later evidence of cardiac lesion is about as likely to appear in cases which at this period show a slow pulse as in those cases with marked acceleration. Of greater importance is the rate of the pulse at subsequent periods. When the temperature falls the pulse, as a rule, returns to the normal rate, but variations are observed. In some cases the rate becomes much diminished, while the temperature is still elevated. Pulses of from 55 to 58 beats a minute are not uncommon, and occasionally the pulse rate falls suddenly to from 20 to 40 beats, so that one may speak of a pulse crisis. This crisis may occur several days after the temperature has reached the normal point; in other cases it falls more gradually. Especially notable at this stage is the instability of the heart's action; daily variations of 20 and more beats may occur. In other cases the pulse rate remains high after the temperature has become normal. Such irregularities may be transient, no further symptoms of circulatory derangement following, but when marked they are usually the precursors of more serious manifestations. The pulse after dropping to normal with the temperature may remain there for several days and then suddenly rise to 130 or more, persisting at this rate with wide daily variations for several weeks. This rise, in what Schmoltz terms the tertiary period, always indicates some cardiac lesion.

During the early stages of the disease the first heart sounds often show some impurity or a systolic murmur, which commonly vanishes in a few days, but in some cases becomes more marked as the temperature falls, while other symptoms arise which leave no doubt as to the presence of a heart lesion. More often murmur develops at the end of the first or during the second stage; not infrequently they appear later. Their character is various, at times blowing, at times harsh and humming; no diagnostic importance can be attached to these differences. They are almost always systolic in time, sometimes louder at the apex, sometimes at the base. Such murmurs are usually accompanied by an accentuated second pulmonic sound. The second pulmonic may be accentuated before the development of a murmur or without a murmur; in these cases it is frequently associated with an irregular or a rapid pulse. Such changes may be present with no alteration in the area of cardiac dulness or in the position of the apex beat, but frequently there is enlargement to the left and often also to the right and upward, while the point of maximum impulse moves outward. In 14 per cent. of his cases Schmoltz observed an increase in the area of dulness; in some of these no murmur developed. It is important to note that such dilatation may be deferred to the period of convalescence, when it may follow suddenly some unusual exertion. Schmoltz finds that dilatation sets in most frequently toward the end of or after the febrile period, sometimes after a considerable length of time. While irregularity of the pulse is usual in these cases, it may be absent, as in diphtheria, even in cases with most marked dilatation.

Practically no subjective symptoms accompany these lesions as long as the child remains in bed, though there may be palpitation and a little dyspnœa. The temperature remains normal or is but slightly elevated. Cardiac complications are not much more frequent after the severer attacks of scarlet fever with marked involvement of the throat than after milder cases, and some of the gravest cardiac lesions follow very mild attacks, with uncomplicated convalescence. As rheumatoid pains are very common in scarlet fever, Heubner has referred both joint and heart lesions to a rheumatic infection. Schmoltz, on the other hand, finds that cardiac lesions are no more frequent among the cases with arthritic pain than in instances without these symptoms. He believes the two complications bear no further relation to one another than that of having a common cause. Hearts damaged before the onset of the infection bear the brunt of the attack well. Of 27 cases showing, on admission, a slight mitral insufficiency, 17 passed through the illness without apparent damage to the heart; in 5 cases there was transient arrhythmia or an unusually rapid pulse in the latter part of the disease; in 3 slight dilatation was made out.

The ultimate outcome of cases with these cardiac manifestations varies. In a large proportion all the symptoms disappear. In 29 cases of this series, however, changes persisted on discharge. In 19 of these cases and 5 more which, on discharge, showed no abnormalities, he was able to observe subsequently. In 18 abnormal cardiac conditions were found. In 16 signs of a definite mitral insufficiency, systolic murmur, accentuated second pulmonic, and in some fairly well-marked cardiac enlargement. In the other 2 cases a systolic murmur alone was found. In 6 cases the heart seemed normal. Of the 18 affected, 5 had become worse after discharge. These displayed during their illness some circu-

latory disturbance, but on discharge were quite free from unusual signs. Three cases had improved after discharge, the symptoms then present having cleared up. Of the 16 cases with persisting cardiac lesions in 13 over six months had elapsed since the attack, in 4 one year, and in 2 over five years.

These heart affections were formerly all referred to an endocarditis, but since Romberg's paper the importance of the myocardial changes has been more generally recognized. Endocarditis is a very rare incident in scarlet fever. In a review of 30 autopsies the author could find only 3 showing slight changes in the valves. Myocarditis is, on the other hand, very common, and there can be little doubt that most of the symptoms are referable to this lesion. Why the signs of a mitral insufficiency should so frequently persist may be variously explained. It is now well known that a relative insufficiency, lasting indefinitely, may depend upon changes in the heart muscle.

The heart in scarlet fever should be carefully watched, not only during the disease, but also in convalescence. Grave results may follow too early strain. When cardiac complications arise, the one important point in treatment is to insist upon prolonged rest in bed.

Diseases Due to Trypanosomes.—ROBERT KOCH (*Deutsche med. Wochenschrift*, 1904, xxx. p., 1705) states that since the discovery of the malarial parasite by Laveran and pyrosoma bigeminum by Theobald Smith, recognition of the importance of the protozoa has spread rapidly. The appreciation of the important role of one especially interesting group of parasites, the trypanosomes, which were shown by Bruce in 1895 to be the cause of the tsetse disease in animals, is a result of investigations of the past few years. The trypanosomes belong to the group of flagellates, and in fresh blood preparations move about with great activity. They are two or three times longer than a red corpuscle, have a long flagellum on the fore end, and are surrounded by a loose, undulating membrane. Stained by the Romanowsky method, they show a large red nucleus and an intensely red centrosome situated near the hinder end, from which a red line runs along the undulating membrane, ending in the red tail. The body is blue and without pigment.

Trypanosome infections have many features in common with malaria. They may run an acute course, but are more usually chronic and sometimes drawn out for years; they cause irregular fever, often with longer or shorter remissions, anæmia, emaciation, loss of strength, localized œdema, enlargement of lymph glands and spleen.

The best known of these infections is the tsetse disease, due to trypanosoma Brucei, and transmitted by the bite of glossina morsitans, the tsetse fly. The disease was first observed in the valleys of the Limpopo and Zambezi, but is now known to extend on the east coast of Africa from Natal to Somaliland and on the west coast up to Senegal, through the Congo basin about Lake Tchad, and upward to Southern Algeria. Nearly all domestic animals are susceptible; the organism is very pathogenic for laboratory animals.

Similar to tsetse disease is surra and the mal de Caderas. Surra is a disease affecting horses and camels in Asia; in some regions it attacks the cattle. It extends from Persia to Indo-China, and in recent years has been conveyed to the Philippines, to Java, to Mauritius, and to

Abyssinia. Mal de Caderas is prevalent among horses in South America, extending from Argentina to the Amazon. Both diseases are conveyed by a fly of the variety of Stomoxys, and the trypanosomes causing them are indistinguishable in their morphology and pathogenicity from the tsetse trypanosome.

These observations became of special interest when Dutton, in 1901, found trypanosomes in the blood of a native of Gambia suffering from what was thought to be malaria. Confirmation of his report soon arrived from all sides, and its significance was fully realized when Bruce, in 1903, established the etiological relation of trypanosomes to the sleeping sickness. This disease was known in the beginning of the last century. It was observed principally among the negro slaves on the west coast of Africa and frequently transported to the West Indies, where it was noted that the disease might occur in slaves from five to eight years after leaving Africa. It never took root in the Indies, evidently, from our present knowledge, because there were no flies to carry it. At that time the affected area in Africa reached from Senegal to the boundary of Angola, but recently it has been carried to the Congo basin, and from there, in 1896, to Uganda. Here the disease seems to have found especially favorable conditions, about 200,000 people having succumbed to it in a few years. Bruce, among 80 apparently healthy natives, found that 23 showed trypanosomes in the blood. The symptoms of human trypanosomiasis are similar to those observed in animals. Sometimes there are no symptoms for a long period after infection; then irregular fever, anæmia, loss of strength, emaciation, local œdema, erythema, glandular enlargements (particularly of the back of the neck), and enlargement of the spleen.

The blood infection—trypanosomiasis—and the sleeping sickness were at first regarded as different diseases, but when it was shown that patients with the sleeping sickness also had trypanosomes in their blood, and that those with trypanosomiasis frequently developed the sleeping sickness, it became evident that the nervous symptoms were due merely to a special localization of the general infection. The organisms are carried by the fly glossina palpalis and are, in all respects, analogous to those discovered in the tsetse disease, surra, and mal de Caderas.

The trypanosomes obtained from cattle in South Africa and described by Theiler in 1902 are easily distinguished, in being from two to three times longer than those from other sources. They are also peculiar in being pathogenic for cattle alone. The disease, although widespread, is mild, and not more than 5 per cent. of affected animals die. The rat trypanosome is also a definite variety. It is characterized morphologically by the drawn-out and pointed character of the hinder end and by the fact that the centrosome is nearer the centre. The infected animals do not appear ill, and the organism cannot be transferred to other species.

The trypanosomes of cattle and rats are distinguished by constant qualities which are lacking in the organisms of tsetse, surra, mal de Caderas, and the sleeping sickness. The former have a characteristic morphology, a uniform virulence, and a single host, qualities pointing to an adaptation to surroundings enjoyed through many generations; the latter are morphologically variable, show a wide range of virulence, and can be transferred to numerous species, qualities pointing to a recent

invasion and an incomplete adaptation to new surroundings. These qualities are well illustrated in the tsetse trypanosome. It is relatively small and has a broad blunt end when grown in rats, cattle, and dogs, while in horses it is larger, the hinder end being drawn out and tapering, so that they resemble somewhat the rat trypanosomes. Of animals passing through a tsetse region, one may acquire an extremely virulent infection and another a very mild infection; and the respective organisms may retain these characters when inoculated into other animals. The virulence of an organism may, however, be greatly increased by successive passages through animals, and it was noted that dogs who resisted inoculation with a trypanosome of low virulence would succumb to the same strain after it had been passed through a number of other dogs. It is also possible by varying the animals to reduce the virulence. While the tsetse trypanosome can be successfully inoculated into nearly all mammals, one meets with varieties which are more virulent for cattle, others for camels, others for horses, etc., indicating a tendency of the parasites to adapt themselves to their host; and such a tendency may, through many generations, become a fixed character.

It has been shown that animals having withstood an infection with a trypanosome of low virulence acquire an immunity against subsequent inoculations with more virulent strains. This fact might suggest a ready means of preventing the disease. Experience, however, has shown that trypanosomes may live in these immunized animals for many years after the protective inoculation, and result in the end in a more fecund source of infection. All investigators consider that antelopes and buffaloes who harbor the organism without suffering themselves are the chief factors in the spread of the disease. Too little is known of the habits and life history of the flies of the Stomoxys and Tabanus species to direct an active campaign against them, but it has been repeatedly noted that with the destruction or emigration of the wild animals both the tsetse fly and the tsetse disease disappear. Drugs have proved of some benefit in human trypanosomiasis, and the results permit the hope that much more effective ones will be discovered.

At present the most fruitful method of attacking the disease is by prevention, and the necessary steps are very simple. All infected animals should be killed, and those suspected carefully isolated. It is important to examine even healthy animals, as it has been found that, without symptoms, they may harbor the organisms for years. The value of these precautions is illustrated in the respective experiences of Java and Mauritius. Surra was brought from India to both countries by cattle. In Java the disease was early recognized and careful isolation practised, so that little damage was done. In Mauritius it was recognized late, and even after recognition little effort made to check it, with the result that in two years all the horses and mules and nearly all the cattle died.

[Those interested in the subject of trypanosomiasis are referred to the admirable work of Laveran and Mesnil, *Trypanosomes et Trypanosomiasis*, 8°, Paris (Masson), 1904, and that of Musgrave and Clegg, *Trypanosoma and Trypanosomiasis, with Special Reference to Surra in the Philippine Islands*, 1903, No. 5, Department of the Interior, Bureau of Government Laboratories, Biological Laboratory, 8°, Manilla.—W. S. T.]

THERAPEUTICS.

UNDER THE CHARGE OF

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Sodium Salicylate in Exophthalmic Goitre. — DR. G. JOUSSEMET states that while this valuable treatment of exophthalmic goitre is by no means new it seems to be little known. It is not applicable to all forms of the disease, but in the pure form, with or without thyroid enlargement, it will be found to be of worth. Treatment of the general condition is a necessary adjuvant of the salicylate. A drachm or more per day may be given in the cures of rapid course, but in those of slower evolution smaller dosage will suffice. The objections to the treatment, since it must be in many cases continued for a year or more, are the possibility of injuring the kidneys—making frequent urine examination necessary—disturbing the digestion and the unpleasant taste of the drug. Sodium bicarbonate may be given as an adjuvant, but by far the best method of administration is to give it largely diluted in water or milk. Some patients who bear the drug particularly ill will tolerate it when given mixed with a large quantity of vichy. Untoward symptoms may necessitate intermission of the treatment. The long continuance of the treatment is its greatest disadvantage, but this drawback exists in other methods as well.—*Revue francaise de médecine et de chirurgie*, 1904, No. 34, p. 811.

The Action of the X-rays upon Leukæmic Blood.—MM. AUBERTIN and BEAUJARD conclude an article upon this subject with the following *résumé:* Radiotherapy provokes in myelogenous leukæmia a diminution of the number of the leukocytes, but this diminution is not regularly progressive. Each application of the rays is followed by a sudden and considerable increase of the white cells, but following this there is a slow but progressive diminution in their number. At first the increase in the cells is almost immediate, but as the organism becomes used to the treatment it becomes more slow. Finally the increase becomes imperceptible and only the diminution is noticeable. The increase in leukocytes involves both the myelocytes and the polynuclear cells, and occurs as a result of a rapid overproduction and maturation—which is an improbable theory—or, as a result of an unknown action of the *x*-ray upon the splenic tissue, causing an emigration of adult leukocytes just as they normally emigrate from the bone-marrow. In fine, the first effects of radiotherapy are these profound

changes in the blood taking place before the spleen has begun to appreciably diminish in volume.—*La presse médicale*, 1904, No. 67, p. 533.

Bioson.—Dr. M. Heim reports on this albuminate of iron-lecithin combination as follows: Bioson combines the nutrient and blood-enriching properties of iron and the nerve-strengthening action of lecithin; it may be administered for long periods without disturbing digestion; it is well borne in acute febrile conditions; it increases body weight; it increases the appetite; it augments the hæmoglobin and the number of red blood cells; it acts well in exhausting diseases, and is recommended by its comparative cheapness.—*Berliner klinische Wochenschrift*, 1904, No. 22, p. 593.

Thiocol in Tuberculous Enteritis.—Dr. A. L. Vignon cites a case of pulmonary tuberculosis in which this drug was used with excellent effect. From the beginning it caused an increase of appetite, a cessation of the night-sweats, a tendency toward healing of the lesions, an improvement of the general condition, and an increase in body weight. When the drug was stopped the symptoms reappeared and in addition the infection spread to the intestine. Thiocol was again prescribed and the symptoms immediately became less distressing. The enteritis and bronchitis disappeared, the appetite returned, and the patient's weight began to increase. Observation of this case seems to show that thiocol acts indirectly upon the pulmonary lesions by causing a remarkable amelioration of the patient's general state. The author believes this agent to be the drug of choice in the treatment of tuberculosis, since in every case it stimulates appetite, digestion and assimilation, and in the case cited, at least, it caused a spontaneous arrest of the symptoms of the enteritis, and consequently seems to have an antiseptic action upon the intestinal tract.—*Le progrès médical*, 1904, No. 36, p. 152.

Rectal Feeding, Especially in Gastric Ulcer.—Dr. C. Kleineberger, after investigating Lenharz's statement that blood deterioration follows feeding *per rectum*, and that the anæmia secondary to the hæmatemesis is augmented, concludes that in the course of rectal alimentation the composition of the blood is not affected, nor is there deterioration of the heart's action. The hæmoglobin content of the blood is not lowered unless further vomiting of blood takes place. The author's treatment was as follows: Complete cessation of feeding by mouth for from nine to twenty days after hemorrhage; rest in bed for at least three weeks; two or three nutritive enemata daily, consisting of from 6 to 12 ounces of milk, 2 or 3 eggs, a coffeespoonful of salt, and a dessertspoonful each of sugar and plasmon. In patients whose rectum was irritable, a few drops of laudanum were added. An hour before the feeding the bowel was irrigated with a quart of water.—*Therapie der Gegenwart*, 1904, No. 5, p. 197.

The Treatment of Coma.—Dr. Fiessinger finds that in meningeal affections complicated by coma lumbar puncture is attended with good results. In coma due to cerebral hemorrhage he employs the usual methods, and when he bleeds does so with great care. As a

prophylactic measure against the coma of grip or acute mania he advises warm baths lasting in the former affection fifteen minutes to three hours, in the latter one to two hours, morning and evening. In the coma of sunstroke he prescribes the application of ice-bags to the head and venesection. In coma brought on by intense cold he rubs vigorously with snow or cold water and gives cold drinks. Hot drinks are indicated after consciousness has been restored. Diabetic coma is to be treated by prophylactic measures. Coma occurring in the course of the infectious diseases is variously treated. In rheumatism intravenous injections of sodium salicylate and cold baths are recommended. In other infections hydrotherapy by means of cold or warm baths meets the indication.—*Journal des praticiens*, 1904, No. 28, p. 437.

Subcutaneous Injections of Air in Neuralgias.—Drs. Mongour and Carles employ the following technique: Under antiseptic precautions and with care not to draw even a drop of blood, a needle is inserted into the intramuscular planes of the chosen site, and a bicycle pump or bulb of a Pacquelin cautery being attached, the desired quantity of air is injected. This varies from one-half pint to one pint, depending on the sensations of the patient. The injection of the air has in some cases an immediate anæsthetic effect. Light massage should follow the procedure, and it is well to repeat this for a few days until the crepitation due to the air has disappeared. Of 25 cases of sciatica treated by this method, 13 were cured and but 2 were unimproved. The explanation that this method of treatment acts by mechanical pressure upon the nerve endings seems to be the true one, since the massage after injection increases the good effect of the procedure, and since the pain reappeared to some extent as the air is absorbed, only to disappear after a renewal of the treatment. Consequently air is as effectual an injection as any of the other gases with which experiments have been made. The treatment is applicable to various forms of neuralgia which resist other methods.—*Journal de médecine de Bordeaux*, 1904, No. 35, p. 633.

The Treatment of Whooping-cough.—Dr. Stepp believes that fluoroform has a specific action in pertussis. He has obtained in 20 instances what he considers a cure, in that the patients after from six to thirty-seven days of treatment were permanently relieved of the characteristic cough. Fluoroform may be given to patients of all ages without regard to their condition in other respects, which is a strong point in its favor. The paroxysms are diminished in force and frequency from the first day of its administration. The drug is given in 2 to 2.5 per cent. aqueous solution, which possesses no taste or odor, and has the single disadvantage of causing slight pharyngeal irritation as it is swallowed. To young infants a coffeespoonful may be given every hour; to older children, from a teaspoonful to a dessertspoonful.—*Therapeutische Monatshefte*, 1904, No. 11, p. 549.

Arsenic and Creosote Treatment of Pulmonary Tuberculosis Compared.—Dr. Ducot contrasts sodium cacodylate given hypodermically with thiocol in pulmonary tuberculosis as follows: The use of the cacodylate increases the appetite and the body weight; these effects are temporary

and due to a direct action of the drug upon the anatomical elements of the body. The toxicity of the cacodylate forbids its continued use, and the drug is contraindicated in fever and hæmoptysis and does not seem to influence the pulmonary lesions. In the author's experience, on the other hand, thiocol is without poisonous effects and does not disturb the digestion. It is a stomachic and an intestinal antiseptic, it increases the appetite and the bodily vigor, it reduces fever, and causes the functional symptoms to disappear, and, more than all these, it tends to cicatrize the pulmonary lesions. Its only disadvantage is that it tends, on account of its ability to produce congestion, to cause the expectoration of bloody sputum. With the cacodylate we get an antianæmic effect; in thiocol we have a therapeutic agent of true antibacillary power.—*Gazette médicale de Paris*, 1904, No. 47, p. 528.

Landerer's Treatment of Pulmonary Tuberculosis.—Dr. F. Schrage describes the mode of action of this method of treatment as follows: Its objects are (1) to produce a general leukocytosis; (2) to produce an aseptic inflammation about the tuberculous foci. In 1893 Landerer introduced the treatment by hetol, an easily soluble preparation of sodium cinnamate. The treatment is most applicable to incipient and uncomplicated cases—*i. e.*, patients displaying no rise of temperature and with slight destruction of lung tissue. On the other hand, apyretic cases with small cavities are also amenable. Rapidly progressing and advanced cases should be treated only in sanatoria. In properly selected cases the cinnamate seems to have a true curative action; following the injections the evening rise of temperature disappears, the night-sweats are quickly stopped, and the weight increases. The rales disappear; the sputum is less and becomes free from bacilli. The efficacy of this treatment should, in the author's opinion, encourage us to make the diagnosis as early in the disease as possible.—*Münchener medizinische Wochenschrift*, 1904, No. 44, p. 1954.

Eumydrin, a New Drug for Diminishing Sweating.—Dr. Bernard Engländer states that this agent, which is methylatropine nitrate, is an excellent preventive of excessive sweating, especially in pulmonary tuberculosis. It is a white powder, moderately soluble in water, and may be given in doses of from 0.015 to 0.0375 grains. Most patients bear the drug well, and there is seldom any complaint of gastric distress, ringing in the ears, dryness of the mouth, palpitation, or insomnia. Increased intestinal peristalsis and pupillary dilatation may occur. In addition to checking the perspiration in tuberculous conditions, the general condition of the patient is improved, the appetite is increased, and his bodily vigor is augmented.—*Weiner klinisch-therapeutische Wochenschrift*, 1904, No. 48, p. 1261.

The Treatment of Glycosuria and Diabetes.—M. M. Huchard and Fiessinger consider the arsenical mineral waters useful in pancreatic diabetes, with antipyrin or aspirin as adjuvants. Nervous diabetes may respond to the use of potassium bromide in dosage of 1 drachm daily or valerian in the form of the extract. In all forms of the disease the diet is the same. Potatoes (4 to 5 ounces per day) are allowable. The various forms of diabetic bread, except possibly the aleuronat bread of Ebstein, all have their disadvantages. Milk diet may be

employed in hepatic diabetes with an enlarged liver, and this regimen is necessary if the condition is complicated with intestinal nephritis. The patient should drink considerable amounts of the infusions of quassia or cinchona; only a little wine should be permitted; beer, cider, and sweet wines are to be interdicted. Fruits, preferably apples and peaches, may be eaten in small quantity. Starchy and sugary foods must be suppressed. Meats except liver may be allowed in moderate amount. Eggs are excellent, as also are all the green vegetables. With regard to drugs, antipyrin, aspirin, and arsenic are the most useful. The first should be given in doses of 20 grains in two powders before the principal meal for about a week, and followed by sodium arsenate $\frac{1}{20}$ to $\frac{1}{15}$ of a grain. If the glycosuria is marked and the digestion good, a pill containing the latter drug and codeine may be given. To this pill valerian may be added to lessen the polyuria. Moderate exercise, warm baths, and avoidance of emotional disturbance are to be advised.—*Journal des praticiens*, 1904, No. 43, p. 691.

Resorcin in Sycosis.—M. G. BELGODÈRE reports upon the results attained by the use of a spray of 1 per cent. aqueous solution of resorcin, and attributes excellent results to its employment. The solution is used in an atomizer, the nozzle of which is held eight to ten inches from the affected part, and is employed for from ten to twenty minutes daily. The spray passes easily through the hair and penetrates to the skin. It is often useless to shave the region or to epilate in the milder cases, and it is unnecessary to apply wet dressings of the solution, except in severe cases where they may be applied during the night. In the author's experience, under this treatment, the redness decreases, induration disappears, and the tissues regain their normal condition, the cure necessitating a period of from two to seventeen weeks.—*La semaine médicale*, 1904, No. 48, p. 392.

OBSTETRICS.

UNDER THE CHARGE OF

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Tubal Gestation.—In the *Journal of Obstetrics of the British Empire*, February, 1905, BERKELEY and BONNEY contribute a paper entitled "Tubal Gestation."

They have examined 18 specimens of early tubal pregnancy, the period varying from nineteen to thirty days. They found that early tubal gestation is entirely intramural. In no point did the gestation sac communicate with the lumen of the tube.

So far as the formation of a decidua was concerned in the specimens examined, there was an absence of connective-tissue reaction to the

invading ovum. They did not find decidua cells in the ovary of the opposite tube or the peritoneum.

Their specimens showed that the ovum travels toward that part of the tube where the nutrition and the vascular supply is most copious. In most cases implantation occurred in the attacked half of the tube wall.

The writers believe that the cells previously described as decidua serotina are identical with the trophoblast of the early gestation sac. The splitting of the circular from the longitudinal muscle coat was very striking in the specimens studied by the writers.

In these specimens syncytium was found in masses on free surfaces in contact with maternal blood. Fetal tissue invaded the walls of the bloodvessels from without, or sometimes replaced part of the vessel wall. In the latter the tissue probably made its way along the lymphatics.

The rupture of the primary gestation sac may be brought about by mechanical causes or by the destructive action of the trophoblast.

Another factor is bleeding from maternal vessels into the gestation sac, causing a sudden rise in tension, rupturing the sac wall already weakened by the action of the trophoblast. This hemorrhage either occurs into the intervillous space containing blood or into the space between the amnion and chorion. In most of the cases examined there was undoubted evidence of bleeding into the sac.

Rupture usually takes place in one of three directions: first, the gestation sac may rupture through the tube into the peritoneum or into the broad ligament; this occurs most frequently at the isthmus of the tube, and results from the destructive action of the trophoblast alone.

The gestation sac may also rupture into the lumen of the tube; this constitutes tubal abortion.

The gestation sac may also rupture within the wall of the tube, constituting intramural rupture.

In these cases the longitudinal muscle coat is split off from the circular muscle coat and blood extravasates between these two. A mass forms which appears externally as a sausage-shaped enlargement of the tube.

These cases are mistaken for hæmatosalpinx, which they resemble.¶

This blood sac frequently bursts, and the contents then escape into the peritoneal cavity, broad ligament, or lumen of the tube. The first is the most common perforation, which is often closed temporarily by blood clot. The blood sac often bursts into the tube itself, and blood may pass thus into the peritoneum or may accumulate into the tube.

Intramural rupture is more common in the ampulla of the tube than in the isthmus.

Combined varieties of the rupture of the gestation sac, also, frequently occur.

So far as the causation of extrauterine pregnancy is concerned, the examination of these specimens gave no information.

There was no sign of pre-existing salpingitis, nor were there abnormalities in the tubes.

In 3 pregnant tubes of less than a month's duration, the corpus luteum was found in the ovary of the opposite side in 2 cases, and in the ovary of the same side in 1 case.

An Investigation into the Causation of Puerperal Infections.—FOULERTON and BONNEY (*Journal of Obstetrics of the British Empire*, February, 1905) give the results of their investigations in 96 cases, in which 12 had a normal puerperal period, 54 had either miscarriage or labor at term, followed by fever, and 30 cases had cervicitis in non-pregnant conditions.

Fourteen of these patients were in a lying-in hospital, while the remainder came from out-patient departments and private practice.

Material for examination was obtained by using a glass tube, the shape and dimensions of an intrauterine douche tube. This served for a sheath for a wire with a plug of sterilized cotton-wool at its extremity. The upper end of the glass tube was covered with a cap of cotton-wool. After sterilization the cap of cotton-wool was dipped in a mixture of melted paraffin and thymol. After the glass tube had been introduced into the lower part of the uterus, the cotton-wool cap was withdrawn by a piece of string, the wire carrying the sterilized plug of wool was pushed up to the fundus, and a sample of the uterine contents was obtained. The cultures were then made.

These investigations are especially valuable, because they represent the average case seen by physicians, both in hospitals and in private houses.

In the normal puerperal period the cavity of the uterus was sterile.

In 54 cases there was fever in the puerperal period; 48 of these were after labor at full term, and 6 after primary labor or miscarriage.

In 14 of these cases there were severe symptoms terminating in death, and in all of these bacteria were found within the uterus.

In 26 cases there were severe symptoms terminating in recovery, and in these bacteria were found in the uterus in 21 cases.

In 14 cases there was fever not exceeding 102°, and in these 14 the uterus was sterile in 10, and bacteria were present in 4.

Regarding the kind of germs in fatal cases, the bacteria which seemed most abundant and potent were streptococci and the micrococcus of pneumonia. In many cases these bacteria were associated with others.

In the second group of cases, with severe symptoms terminating in recovery, streptococci predominated, but the micrococcus of pneumonia was also found. It seemed as frequent and as potent as the colon bacillus in the last group of cases, in which the fever was slight and the uterus was sterile in 10 out of 14. In the 4 cases in which germs were found within the uterus, the streptococcus was absent. The bacteria present were the staphylococcus pyogenes albus and bacillus coli communis.

In non-pregnant women suffering from cervical inflammation, 30 in number, cultures of bacteria were obtained in 16. These were the staphylococcus pyogenes aureus, micrococcus of gonorrhœa, and the bacillus coli communis.

The general result of the bacteriological work in these cases was that streptococci were present in the uterus in 25 out of 39 cases of infection in which cultures from the uterus were taken.

In 17 cases where the contents of the uterus were apparently sterile, streptococci were found in the vagina in 2 cases.

In severe fever, streptococci were present in the uterus in 25 out of 40, and in the vaginal secretion in 2 out of 5.

In 15 cases in which the contents of the uterus were sterile, a marked proportion were primiparæ, with considerable lacerations of the cervix

or perineum. A noticeable feature was the absence of the micrococcus of gonorrhœa and anäerobic organisms.

In non-pregnant cases, streptococci were not found. Another noteworthy fact was the apparent absence of the bacillus coli communis in cases of primary infection.

The writers believe that puerperal fever may be caused by primary infection of lacerations in the vaginal walls or perineum, with sometimes secondary infections of the uterus. There may also be primary infections of the contents of the uterus or at the placental site.

So far as the treatment is concerned, the writers believe that when bacteriological proof of the nature of the infection is wanting, a case of puerperal fever should be treated as being due to streptococci infection.

Active curetting of the uterus in puerperal infection is not advisable. They believe that the best treatment for streptococci infection is the administration of serum, with thorough cleansing of the uterus cavity, followed by intrauterine douches.

In cases of streptococci infection the fever is often transient, subsiding within forty-eight hours, without treatment.

Autogenetic infection is probably, sometimes, the cause of fever, but the use of vaginal douching before labor is not advisable.

In the cases reported the temperature rose with severe symptoms when bacteria entered the uterus. In cases having severe lacerations of the cervix or perineum, the symptoms were much less severe, and the temperature did not rise above 102°.

Artificial Termination of Pregnancy by the Use of Bougies.—JACOBY (*Archiv f. Gyn.*, 1904, Bd. lxxiv., Heft 2) gives the results of 228 cases of induced labor by bougies in the Manheim clinic.

There were 81 cases of contracted pelvis. In these the fetal mortality varied directly with the degree of pelvic contraction and with the shortness of gestation. Two facts are clearly brought out: first, that labor should not be induced in contracted pelves in the interest of the child before thirty-five weeks of gestation, and, second, the fact that in these cases operative treatment during labor largely increases fetal mortality.

As to the percentage of children surviving labor in contracted pelves, it is stated as 65. Evidently the results of induced labor in contracted pelves, so far as the children go, are much inferior to those of Cæsarean section.

So far as the mothers are concerned, in 50 cases, 3 died; 1 from hemorrhage, and 2 from septic infection.

This causes a maternal mortality, in 50 cases, of 6 per cent.

In 57 cases labor was induced by bougies for maternal disease, for eclampsia, nephritis, heart disease, mental disease, pernicious nausea, uterine displacement, polyhydramnios, and fetal death. Bougies alone were used and bags were not employed. The average time in which bougies remained in the uterus was twenty-four hours.

In a case of polyhydramnios, a bougie was left fifty-four hours in the uterus. The patient died after labor.

If those cases were removed in which the maternal disease influenced largely the condition, the morbidity rate would be between 8 per cent. and 9 per cent.

Hemorrhages in Eclampsia.—Lobenstine (The American Journal of the Medical Sciences, February, 1905), at the Sloane Maternity Hospital, found, in 12,000 deliveries, 152 cases of eclampsia. Among these 7 had hemorrhages and 5 occurred during the past two years.

In Case 1, on the second day after delivery, the patient was delirious, without convulsions, but had severe abdominal pain and distention. There was great tenderness over the liver. Toward the middle of the day the patient began to vomit coffee-ground material, speedily followed by death.

In Case 2, on the third day of the puerperal period jaundice, pain and tenderness over the liver and vomiting of coffee-ground material occurred. This continued until the fifth day of the puerperal period, after which the patient speedily improved, and finally recovered.

In Case 3, a white primipara, the same symptoms began soon after delivery. The patient died on the fourth day of the puerperal period.

In Case 4 the patient was admitted in an unconscious condition, having had several convulsions. On the first day of the puerperal period she became jaundiced, with distended and painful abdomen and a very high leukocytosis. Her temperature rose to 102.8°. On the second day of the puerperal period her condition suddenly grew much worse. There was marked tenderness over the liver, bloody vomit, and tarry stools. In the early afternoon she vomited bright-red blood and soon after died.

Case 5 was a woman in the second pregnancy, six and a half months advanced, who came into the hospital complaining of headache and dizziness. The uterus was emptied, and on the first day of the puerperal period coffee-ground vomiting, with abdominal pain and distention, occurred. On the second day the patient was delirious, jaundiced, vomited coffee-ground material, and had subcutaneous hemorrhage. She vomited bright-red blood at times, gradually became comatose, had frequent, loose, dark, tarry stools, and died on the eleventh day.

In Case 6 the patient passed through nine days of the same symptoms, and finally made a gradual recovery. The mortality of these cases was 70 per cent. There is no record of autopsies performed upon any one of them, although autopsies are quoted as reported by other observers. Clinically, the significant fact about these cases was the very evident profound toxicity and the severe involvement of the liver. The significance of hemorrhage after eclampsia may be inferred from the fact that the mortality of the general series of eclampsia cases during the past four years at the Sloane Maternity Hospital was 17 per cent., while in the cases associated with hemorrhage the mortality was 70 per cent.

The writer has several times seen hemorrhage in toxæmia and eclampsia. Recently he had occasion to dilate the uterus by Bossi's dilator and terminate an early pregnancy in eclampsia. The patient did well for the first week, the urine becoming almost normal and the temperature remaining normal. At the end of the first week she had a severe uterine hemorrhage. Symptoms of rapid disintegration of the blood developed, and she died within twelve hours of the occurrence of the hemorrhage.

In infants born from eclamptic mothers, hemorrhage is not infrequently observed about the umbilicus or beneath the skin. Dark, tarry stools and profound jaundice accompany these phenomena in fatal cases.

Concerning Uterine Structure.—STEPHENSON (*Scottish Medical and Surgical Journal*, January, 1905) draws attention to what he considers an erroneous idea concerning the arrangement of the tissues of the uterus. He believes that the common teaching that the muscle of the uterus occurs n distinct layers and that these layers can be demonstrated by dissection and found to extend in definite directions is to be rejected.

He quotes other observers, and adds his own investigations to prove that the walls of the uterus are arranged first upon a connective-tissue framework. The meshes of this framework are flattened in a plane parallel with the surface, being fil ed in with muscular tissue, which appears as plates and not as bundles of fibres when the uterine wall is studied in sections. This lamellate arrangement allows one stratum to move upon another or several to join in a sliding movement in the same direction.

Regarding the lower uterine segment, he believes it is not elastic only, but that it has sufficient muscular tissue to exercise some force. The thickening when the uterus is empty is on the inner side, and there is evidently a difference in behavior between the outer and inner portions of the walls. While he believes that the cervix remains such throughout pregnancy and parturition, he also believes that there is an external muscular wall, uniform in structure, continuous throughout the uterus, and prolonged into the wall of the vagina. He believes that the connective tissue of the cervix is an extension of that found in the body of the uterus, and that it exists in the form of a cone, the base of which is at the vaginal portion of the cervix, the tissue growing thin as it passes upward to the internal os.

During labor the lamellate construction of the uterus appears throughout its whole muscular tissue. When retraction has taken place, this lamellate appearance is fairly well maintained in the outer subperitoneal portion, which is undisturbed in the lower uterine segment, and so long as the placenta is attached it remains at the placental site. From the retraction ring upward the lamellæ of the inner portion are crumpled into a confused mass, where islets of muscle, surrounded by connective tissue, can be demonstrated.

The ovum is retained within the uterus, not by the act on of sphincter fibres, but because the ovum is an integral part of the pregnant uterus, and the shape of the womb and its structure further its retention. During pregnancy the cavity of the uterus is kept closed by the cervix, which resists strain brought to bear upon it. When the cervix softens and the womb opens, the ovum may be expelled.

As regards the action of the uterus in labor, he believes tha' the preliminary softening and shortening of the cervix, and even its disappearance, should be considered a process of dehiscence. This botanical term, originally applied to changes in the connection between ripe fruit and the parent stem, accurately describes this process in the uterus.

On the contrary, in labor the whole uterine muscle is thrown into action, steadily progressing with increasing energy, often through hours. On the other hand, the process of dehiscence may extend over days and weeks.

GYNECOLOGY.

UNDER THE CHARGE OF

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ASSISTED BY

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Vaginal Ovariotomy during Pregnancy.—MAINZER (*Zentralblatt f. Gynäkologie*, No. 48, 1904) reports the following cases:

Case I. The patient, aged twenty-five years, who was in the fourth month of pregnancy, had an ovarian dermoid the size of the fist. The patient had a normal convalescence, sitting up on the tenth day. The pregnancy was not interrupted.

Case II. A dermoid, the same size as in the former case, was removed per vaginam from a patient two months pregnant. She aborted on the twelfth day after operation, which was attributed to the presence of a large tampon that had been inserted for drainage.

Influence of Castration on Metabolism.—ZUNTZ (*Zentralblatt f. Gynäkologie*, 1904, No. 48), from experiments in 4 cases of oöphorectomy, found that removal of the ovaries caused little or no change in metabolism, nor was any marked increase noted after the administration of oöphorin. This result is the reverse of that observed by Lowry and Richter in their experiments upon dogs, although the same quantities of the drug were administered. The writer infers that there may be a difference in the tablets, whether prepared from the corpus luteum or from the stroma. It is possible also that the age of the ovaries makes some difference in the effect of the preparation.

Migration of the Ovum.—BURCKHARD (*Zeitschrift f. Geb. u. Gyn.*, Band lii., Heft 3) denies the possibility of internal migration because of the size of the impregnated ovum and the direction of the ciliary motion. He is also skeptical with regard to the occurrence of external migration on account of the wide separation of the distal ends of the tubes, and the tendency of the peristaltic movements of the intestines to carry the ovum in a different direction. However, in case both tubes are prolapsed into Douglas' pouch their fimbriated ends may lie so close together that external migration is possible.

Closure of the Tubes.—OPITZ (*Zeitschrift f. Geb. u. Gyn.*, Band lii., Heft 3) calls attention to the fact that there is a layer of loose connective tissue beneath the peritoneal covering of the tube, the peritoneum being closely attached behind the fimbriæ so as to form a ring at this point. When the tube becomes swollen from inflammation the fimbriæ are drawn into the distal opening, which is subsequently closed. The more the tube enlarges the more the fimbriæ are drawn inward until their peritoneal coats adhere, after which fluid accumulates in the tube.

Conservative Myomectomy.—HENKEL (*Zeitschrift f. Geb. u. Gyn.*, Band lii., Heft 3) reports 40 cases, 3 patients subsequently becoming pregnant, 2 going to full term. The writer thinks that the danger of rupture of the uterus in these cases is purely theoretical. In 5 cases the operation was imperfect, fibroids being left; however, sarcomatous degeneration of such growths is extremely rare.

He prefers the abdominal operation, and removes the entire capsule of the tumor, resecting part of the uterine wall. Comparing the results of his conservative work with those of supravaginal amputation, the writer considers the former preferable.

Cystadenoma of the Uterus.—BANEREISEN (*Beiträge zur Geb. u. Gyn.*, Band ix., Heft 1) removed a uterus in the anterior wall of which was a pedunculated tumor. The pedicle was hollow and communicated with the uterine cavity and the interior of the growth. Anatomically the latter consisted of typical lymphadenoid tissue. The glandular structures were continuous with the glands of the uterine mucosa.

Recurrence after Extirpation of the Cancerous Uterus.—MACKENRODT (*Monatsschrift f. Geb. u. Gyn.*, Band xix., Heft 6) asserts, as the result of his observations in 80 cases, in 95 per cent. only the intrapelvic lymph nodes were affected. Since in at least one-half of all cases of cancer of the uterus, even in the early stage, the glands are diseased, failure to remove them means that recurrence is inevitable. The writer adds that in at least three-fourths of the so-called inoperable cases the deeper nodes are affected late in the course of the disease, so that a radical operation may be possible if not delayed too long.

Cysts of the Mesentery.—HAAS (*Zentralblatt f. Gynäkologie*, 1904, No. 50), in reporting a case, states that the principal diagnostic points are sudden acute pain in the abdomen, associated with the presence of a tense, elastic tumor, with slight mobility and tender on pressure. The tumor has no apparent connection with the pelvic or abdominal viscera except the intestines. A coil of gut can usually be demonstrated to be adherent to the tumor, which moves with it.

Enucleation of the cyst is the best method of treatment, provided that this can be done without too great loss of blood. If a radical operation is impracticable the sac may be stitched in the wound and drained.

Inoperable Cancer of the Uterus.—LICK (*Monatsschrift f. Geb. u. Gyn.*, Band xx., Heft 1 and 2) reports 70 cases treated by curettement, cauterization and subsequent applications of perchloride of iron, with one death following operation.

Hemorrhage and sloughing were eliminated in nearly every instance; 70 per cent. of the patients died during the first year, 9 women lived from one to one and one-half years, 1 survived for two years, and 1 over four years. The average duration of life after operation was 228 days.

Injury to the Rectum during Gynecological Examinations.—KELLY (*Weiner klin. Therapeut. Wochenschrift*, 1904, No. 29) reports 4 cases in which injuries occurred. The cause was probably diminished muscular resistance of the rectal wall due to advanced age.

The treatment advised is laparotomy and suture of the rent, unless the perforation can be reached and repaired by vaginal section. A rectal tube should be introduced and the bowels constipated for five or six days.

In elderly subjects rectal palpation must be practised with care, without keeping the finger too stiff.

Operative Treatment of Climacteric Hemorrhages.—QUEISNER (*Zentralblatt f. Gynäkologie*, 1904, No. 51) describes his method of treating obstinate cases in which ordinary methods fail and hysterectomy is refused. A T-shaped incision is made in the anterior vaginal wall, the bladder is dissected from the uterus, and the vesicouterine pouch is opened. The raw surface on the bladder is covered by the anterior peritoneal flap. The uterus is drawn downward, split in the median line, and both halves are sutured to the edges of the vaginal wound. The uterine mucosa, being then thoroughly exposed, is cauterized with the Paquelin. The after-treatment consists in vaginal douches, the patient being allowed to leave her bed on the fourth day. Two successful cases are reported.

Results of Alexander's Operation.—REIFFERSCHEID (*Archiv f. Gynäkologie*, Band lxxiii., Heft 1) reports 241 cases from the Bonn clinic; 96 were kept under observation, 8 of which were failures. In 18 cases one or both ligaments were either not found or were torn in pulling out. Better results were obtained when the canal was not opened.

Stress is laid on the importance of frequent emptying of the bladder during the first few days after operation.

From experiments on the cadaver, the writer is led to take an opposite position from Doléris and others, who claim that the round ligamant can always be found at the external ring, even in infants and in the aged.

Entrance of Fluids into the Tubes.—THORN (*Zentralblatt für Gynäkologie*, 1894, No. 38), from a series of experiments with the cadaver, as well as clinical observations, arrives at conclusions contrary from Döderlein, who affirmed that fluids injected into the uterine cavity frequently enter the tubes and even escape into the peritoneal cavity. In all the reported fatal cases the writer thinks that there was a strong probability that the uterine wall was perforated. The greatest danger, he believes, in injecting caustics is from necrosis of the wall and entrance of the fluid into the veins, especially after curettement for malignant disease of the endometrium. Hence Braun's syringe is not such a dangerous instrument as has been represented, provided that only a few drops are injected at a time, the chances being a thousand to one that superfluous fluid will escape from the os rather than enter a normal tube.

Injuries to the Genital Tract during Coitus.—SAKS (*Zentralblatt für Gynäkologie*, 1904, No. 38) reports the following cases:

Cases I. and II. Two newly married women had profuse hemorrhage for an entire day following the first coitus from an artery in the ruptured hymen, which was controlled in one case by pressure with

tampons saturated in a 20 per cent. solution of ferripyrin, in the other by a solution of perchloride of iron.

Case III. A multipara, aged twenty-five years, had intercourse in a semiprone position. She was seized with a severe pain in the abdomen followed by profuse bleeding. Examination showed a tear in the right vaginal fornix. The hemorrhage was controlled by a tampon and the patient was removed to the hospital, where it was necessary to give a subcutaneous injection of saline solution. She made a good recovery.

Case IV. A girl, aged twenty-five years, bled for three days after her first coitus so that she fainted in the street and was brought to the hospital. A tear was found in the rectovaginal septum, the hymen being intact. The wound healed in a few days after removal of a tampon which had been placed in the vagina.

Case V. Repeated coitus in a newly married woman was followed by sharp hemorrhage, which was due to a lacerated hymen, and was checked by a solution of the perchloride.

Commenting on these cases, the writer calls attention to the comparative infrequency of these lesions, less than 200 having been recorded. Twenty-two fatal cases have been reported from hemorrhage or infection. In one instance a man lost three wives in succession from laceration of the posterior vaginal fornix during the first marital approach.

The cause is usually some anomaly of the female genitals, or unusual size of the male member, or the deeper lesions of the vagina may be due to unnatural positions during coitus. The writer disagrees with those observers who attribute them to excessive sexual excitement on the part of the female.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

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Purulent Maxillary Sinusitis.—Dr. Liambey (*Annales des maladies de l'oreille, du larynx*, etc., December, 1904) reports a case of maxillary sinusitis in a girl aged seventeen years, with a fistula at the inferior angle of the left orbit, consecutive to an odontoma. The affection was said to be of seven years' duration. The middle meatus contained mucous polypi bathed in pus. Puncture of the inferior meatus and syringing through the cannula discharged fetid pus, both by the nose and by the fistula. The upper left canine tooth was absent, and there was a fistula there from which pus exuded on compression of the gums. The sinus was opened and found full of pus and fungosities. As the breach in the opening was being enlarged, an odontoma was forced out, evidently corresponding to the absent canine tooth. The sinus was faith-

fully curetted, and the patient gradually recovered in a little less than six weeks. This case has been recorded on account of its rarity, the reporter having no knowledge of an analogous case in medical literature.

Sarcoma of the Tonsil Apparently Cured by Exposure to Roentgen Rays.—Dr. Charles Lester Leonard (*American Medicine*, December 3, 1904) reports, among other cases, one of sarcoma of the tonsil apparently cured after a series of treatments, external and internal, twice a week, with a high vacuum, and without injury to either skin or mucous membrane. The patient has since been actively engaged in his insurance business for fourteen months.

Primary Tuberculosis of the Pharyngeal Tonsil.—That the lymphoid tissue at the vault of the pharynx may become the seat of tubercle as well as the tonsil and other lymphoid tissue elsewhere in the throat is well known, but primary tuberculosis is very rare and difficult of substantiation. In the *Medical Record*, October 8, 1904, Dr. Donald M. Barstow refers to a case of "Supposed Primary Tuberculosis of the Pharyngeal Tonsil" in a man, aged twenty-nine years, who had suffered with cough for some two years, and for three or four months with a continuous cold in the head and copious expectoration. Examination of the sputum revealed "innumerable tubercle bacilli and quite a large number of streptococci." Posterior rhinoscopy revealed a large amount of very soft and friable adenoid tissue in the vault of the pharynx. Under cocaine this was removed with little difficulty. Two weeks later examination of the sputum "required long-continued search to find tubercle bacilli, and when found they were either isolated or in small groups;" hence the inference that this might be a case of primary tuberculosis of the pharyngeal tonsil. The patient gained in weight rapidly, and was soon dismissed from supervision.

Foreign Body in the Larynx Removed through the Natural Passages.—The case is reported by Dr. Henry Coussieu (*Annales des maladies de l'oreille, du larynx,*, etc., March, 1904). A nervous lady, an hysterical subject, insisted that she had swallowed a pork bone while taking soup, and that she could feel it in her neck. It could not be detected upon laryngoscopic examination, and there were no physical signs to denote its presence in the air passages. Nearly a month later, on presenting herself with a story of bronchitis, a putrid odor from the mouth and a continuance of the sensation of the bone in her neck, laryngoscopic inspection revealed the bone, sure enough, impacted below the vocal bands, beneath which a thin edge of a flat foreign body could be seen in the middle vertical line. This was readily removed with forceps, and measured 29 mm. in its greatest length and 18 mm. in its greatest width. The case is illustrated with wood-cuts, showing the laryngoscopic image and the manner of extraction.

Live Foreign Bodies in the Larynx.—In an excellent, researchful, though rather prolix article on the course to take in cases of animated foreign bodies in the human larynx (*Revue de hebd. de lar., otol., et rhinol.*, March 12, 1904), Dr. Linaras reviews almost the entire literature of the subject, and mentions, among other personal observations

of cases of leeches in the air passages, two which may be noted here for their singularity. In one the leech was situated at the anterior commissure of the vocal bands subglottically. An appointment was made for the patient to go to a hospital for operation; but in the interim an Arabian acquaintance whom he consulted advised him to gargle with a decoction of fresh tobacco-leaves. This he did, and shortly afterward coughed out the leech without any trouble.

In the other case the leech was found to have left the larynx and to have attached itself to the vault of the pharynx. It eluded the first attempt to catch it with forceps by sliding down the pharynx, but eventually crawled up again, when it was caught and extracted.

Morbid Growth of the Larynx of Traumatic Origin.—Dr. John Edwin Rhodes (*Chicago Medical Recorder*, June, 1904) reports a case of a lad whose neck had been caught between a plank and the rollers of a machine. He spat blood for six hours and became dyspnœic and dysphonic. He gradually improved and left the hospital in three weeks, with subsidence of the acuter symptoms, but with continuance of the hoarseness and difficulty in breathing. Nearly two months later he returned with a small abscess in the median line over the thyroid cartilage, which, on opening, revealed the presence of a grape-seed, which had probably made its way there from the œsophagus.

Laryngoscopic exploration revealed marked thickening of the ventricular bands and of the arytenoids, with such deformity of structure as to reduce the calibre of the larynx to scarcely one-fourth of its normal size. From the right ventricular band there projected a mass of tissue the size of a small bean, which acted as a valve in closing the lumen of the larynx during both inspiration and expiration. Attempted removal with crushing forceps having failed, a preliminary tracheotomy was performed a few days later, and, after cocainization of the larynx, the growth was removed with a cold-wire snare without difficulty. At the time of the report the patient's breathing was easy and the voice fairly good, although the lumen of the larynx remained much diminished by the distortion which had followed the accident.

Double Abductor Paralysis of Traumatic Origin.—Dr. Lambert Lack recently exhibited to the Laryngological Society of London (*Journal of Laryngology, Rhinology, and Otology*, June, 1904) a man who had had complete bilateral paralysis for eighteen months as a result of a throat-cut almost completely dividing the trachea at its junction with the larynx. It was supposed that both recurrent nerves were injured. The patient was wearing a T-shaped tracheotomy tube, as there was a narrowing of the calibre of the windpipe just above the external opening into the trachea large enough to admit the tip of the little finger. The paralysis was complete on the left side, with a slight movement on the right side.

Sir Felix Semon thought it must have been a very extensive cut to have reached both recurrent laryngeal nerves, and that it was not easy to understand why the other neighboring structures should have escaped. In his opinion probably the most plausible explanation was that there had been a good deal of new formation of cicatricial connective tissue in the neighborhood of the recurrent laryngeal nerves which had gradually compressed them.

Hemorrhage after Tonsillotomy.—Dr. Oscar Wilkinson (*Journal of Eye, Ear, and Throat Diseases*, July–August, 1904) reports a case of hemorrhage after tonsillotomy relieved by the use of adrenalin. After a double tonsillotomy under nitrous oxide on a girl, aged seventeen years, there was free hemorrhage, which was controlled by the application of adrenalin solution 1 : 1000 and ice gargles. Profuse hemorrhage recurred in two hours. Applications of adrenalin solution controlled the hemorrhage but for a short time. Ten drops of the solution were then given internally, followed by a similar dose in twenty minutes, after which there was no more bleeding.

Prevention of Postoperative Hemorrhage in Intranasal Surgery.—In a paper on "The Collodium Dressing for Intranasal Surgery," presented by Dr. Charles W. Richardson, at the Ninth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology, held at Denver, August, 1904 (*The Laryngoscope*, September, 1904), attention is called to an article by Dr. Kasper Pischal, of San Francisco, on "Collodium after Nose Operation," published in the *Archives of Otology*, vol. xxxi., 1902, and which has received but scant notice from the profession. Dr. Richardson has modified Dr. Pischal's method, and finds that if collodium is properly applied to the whole wounded surface it will absolutely control postoperative bleeding.

Fatalities in Operations upon the Nose and Throat.—At the annual meeting of the American Laryngological Association, held at Atlantic City, in June, 1904, Dr. Francis R. Packard presented a study of the fatal results of operations upon the nose and throat (*The Laryngoscope*, September, 1904), including and supplementing tables compiled by Dr. Holloway in 1896 and Dr. F. W. Hinkle in 1898. Three tables have been prepared, showing 26 deaths attributable to a general anæsthetic, 22 deaths resulting from operations in which the fatal issue was not the result of the anæsthetic, and 5 deaths following operations in which the exact cause of the fatal issue was not given. The total number of deaths attributable to a general anæsthetic was 26: 24 from chloroform, 1 from chloroform and A. C. E. mixture, and 1 from ether. There were 14 deaths from hemorrhage following the removal of tonsils or adenoids, 11 in males, 2 in females; and the sex of 1 case was unmentioned. One death from sepsis and exhaustion is reported following tonsillotomy in a boy. Of the remaining 7 deaths by meningitis, 2 followed an attempt at the correction of a septal deformity; 1 followed an application of perchloride of iron for epistaxis; 1 followed an operation by external access for removal of nasal polypi and orbital tumor (death from purulent leptomeningitis); 1 followed cauterization of middle turbinal with the electric cautery; 1 followed probing the frontal sinus and injection of the lacrymal canal, and 1 followed removal of exostosis.

Of the 5 cases in which Dr. Packard was unable to ascertain the exact cause of death, 1 death was attributable to each of the following operative procedures:

Curettement of nasal polypi.

Galvanocauterization of the middle turbinates, followed by hemorrhage requiring tampon. Death in three days.

Galvanocauterization for bony and membranous occlusion of the right nasal fossa. Death in six days after operation.

Curettement for chronic purulent rhinitis. Death in three days.

Removal of polypi with snare. Empyema of antrum of Highmore. Death eleven days after operation.

In each of these instances death occurred a number of days after the operation, and the history of the cases leads to the inference that it was due to meningitis.

The author of the paper concludes that from the above it is readily inferred that meningitis is a complication of intranasal operation and should not be lightly regarded. It has occurred and proved fatal in enough instances to make us realize that the close relationship existing between intranasal structures and the meninges is not to be overlooked in our intranasal operative work.

OTOLOGY.

UNDER THE CHARGE OF

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Three Cases of Labyrinth Trephining.—BOTEY states that the surgery of the internal ear is still in its early stages, and that excision of that part of the labyrinth accessible through a large opening into the middle ear has but recently been definitely done with success, warrants a more exhaustive notice of the author's paper than would ordinarily be accorded within the limits of a review, but it will still be necessary to refer the reader to the original for the technique of the several operations, the very careful and minute observations made, and the complete reports of the cases.

In all of the operations the primary postaural openings were made sufficiently large to afford free access to and inspection of the inner tympanic and antral walls, and the operation upon the petrous portion of the temporal bone was done by means of the gouge and curette, supplemented by small electric burrs.

The first case was that of a man, aged twenty-six years, who had suffered from a suppurative process in the left middle ear for twenty years, with recently resultant severe occipital pain, excessive tinnitus, and violent attacks of vertigo; there was caries of the epitympanum and antrum, accompanied by labyrinth suppuration, and the operation, which included an extensive incision of the mastoid and middle ear and opening of the external semicircular canal and first whorl of the cochlea, resulted in recovery.

The second case was that of a boy, aged fourteen years, who, following scarlet fever in his second year, had persistent suppurative discharge from the left ear, with occasional exacerbations and symptoms

of pus retention; latterly there had been attacks of vertigo and vomiting; there was destruction of the pars flaccida, cholesteatomatous accumulation in the epitympanum, and antrum and labyrinth suppuration.

The operation consisted of a mastoid exenteration with a large opening into the external semicircular canal and cochlea, the latter being effected by removal of the inner tympanic wall within a line drawn from the posterior end of the oval window to the posterior end of the round window; then, in a curve forward and downward from the anterior edge of the oval window, and then posteriorly in a curve downward to within two millimetres of the tympanic floor and upward to the round window, the opening into the semicircular canal being made with burrs. In this case also a cure was effected.

The third case was that of a man, aged thirty-five years, having a history of bilateral middle-ear suppuration since infancy, with occasional congestive exacerbations in the left ear, accompanied by deep-seated pain extending into the neck and head, but unaccompanied by tinnitus, vertigo, or vomiting. In spite of feebleness his gait was normal, and the tests of von Stein indicated no change in the static and dynamic function of the labyrinth. There was absolute loss of hearing in the left ear, slight dysphagia, and paræsthesia of the left side of the palate with paralysis of the left vocal cord, probably due to lesion of the vagus at the point of its exit.

In addition to the chronic bilateral middle-ear suppuration and necrosis of the left labyrinth, there was a cerebral and cerebellar abscess.

There were two operations: the first a tympanomastoid exenteration with opening of the middle cerebral fossa, and the second, an opening of the posterior cerebral fossa with extraction of the petrous portion of the temporal bone to the depth of the internal auditory canal, and death from shock.

The existence of large openings into the labyrinth, as evidenced in the examination of these three cases, is demonstration of the ease with which pus may enter that cavity in persistent suppuration of the middle ear, especially where there is cholesteatomatous blocking of the epitympanum and antrum, and the cases are also evidence of the fact that operation upon the labyrinth, by way of the epitympanum and antrum, with large opening of the antrum and mastoid, is easily possible.

The fact of the breaking of two burrs inside the external semicircular canal, in the second case, is a demonstration of the extraordinary hardness of the labyrinthine capsule in cases of otorrhœa with osteosclerosis, and at the same time suggests the necessity of using burrs with relatively large shanks.

In the opinion of the author the existence of granulations emerging from a fistulous tract, or from one of the windows, confirms the diagnosis of labyrinth suppuration, especially when the otorrhœa is accompanied by violent attacks of vertigo, vomiting, and disturbances of equilibrium.

Tinnitus, as a rule, does not occur in suppuration of the labyrinth; it is often wanting; when it appears it lasts but a short time, and it occurs only at the beginning of the pyolabyrinthitis.

When a patient in the course of a chronic otitis media is suddenly attacked with vomiting and dizziness, with great inco-ordination of movement, notwithstanding their violence and tenacity, it is not neces-

sary to infer always that pus has penetrated the semicircular canal or the vestibule. It may happen that pyolabyrinthitis does not exist, and that the labyrinthine symptoms are due to simple peripheral compression of the neuroepithelial terminations of the auditory nerve in the ampullæ and the membranous vestibule, occasioned by the presence of cholesteatomatous masses lodged in the attic and aditus, which, by their volume and eccentric growth, push from without inward upon the perilymph and endolymph. Vertigo also may be due to congestion extending from the middle to the inner ear without the entrance of pus or pyogenic germs.

The author cites cases of otorrhœa with dizziness and violent vomitings of long duration, which, despite these symptoms, had no pus in the labyrinth, and in one operative case he found caries and necrosis of the inner wall of the middle ear with absolute integrity of the labyrinthine membrane. In two of these cases, after radical operation with slight curetting of the granulations and removal of small sequestræ, the patients recovered, the tuning-fork and the tests of von Stein showing that the acoustic nerve, on the side operated, effected perfect coordination of voluntary movements.

The third case demonstrates clearly that a pyolabyrinthitis may exist without tinnitus, vertigo, and other disturbances of equilibrium, which reduces the diagnosis to the category of simple surmises, except for the perceived symptoms of suppuration of the labyrinth. This case also shows the possibility of Chaput's operation (resection of the petrous bone), especially when the petrous bone is necrosed, without reference to the existence of the internal carotid artery at the superior part, the sequestræ usually being easily mobilized.

In practice it is rarely necessary to remove so great a part of the petrous bone, Chaput never having done so complete an operation on the living subject.

It is nearly impossible to avoid facial paralysis in resecting the petrous bone, as it is necessary to cut the facial nerve to remove the labyrinth.

The conclusions to be drawn from these three cases and from the author's experience in cases presenting labyrinthine symptoms with suppuration of the middle ear, are given by him as follows:

1. In pyolabyrinthitis cochlear disturbances of the acoustic nerve, such as tinnitus and subjective noises, are very inconstant, quite early, and always temporary. The noises are possibly due to the inflammatory participation of the organ of Corti, the peripheral neurons being more easily destroyed by suppuration than the neuroepithelial terminations of the auditory nerve in the vestibule and in the semicircular canals.

2. Taking into account the small volume of the membranous labyrinth, suppuration nearly always extends over the entire surface, by rapid diffusion of pus, to the whole cochlea, and deafness is complete. This statement the author maintains on his experience with these three cases, and in spite of the contrary opinion of Jansen and Hinsberg, who believe that suppuration frequently affects a part of the labyrinth, principally the external semicircular canal.

3. The most minute examination of the ear, and at the same time the study of the progress of the disease and the appearance of the most characteristic disturbances of equilibrium, do not indicate, in advance, with certainty, the existence of pus in the labyrinth. Only during operation can it be ascertained whether a pyolabyrinthitis really exists

or whether the lesion of the inner wall of the middle ear communicates with the labyrinth.

4. The communications of the middle ear with the internal ear in suppuration are nearly always very small; they can consequently easily be passed unperceived if they are not carefully sought for with a probe.

5. Opening the whole labyrinth with extraction of the promontory, from the oval window to the round window, to the floor of the middle ear, and to the neighborhood of the Eustachian tube, according to the technique advised, ought, in the author's opinion, to be carried out on all occasions, even in cases where the pus is only found in the interior of the external semicircular canal. Not only is the drainage easier, but the pus cannot spread to the inner nooks of the labyrinth.

6. In doubtful cases where some auditory perception remains in the diseased ear, it is better to do a tympanomastoid exenteration and wait for four or five days. If, at the termination of this time, labyrinthine disturbances persist with the same intensity, it is permissible to open the labyrinth by means of a second operation, for an infection is sure to be found.

7. Nystagmus, which is often observed in accidental lesions of the horizontal semicircular canal and in all cases of subacute irritation of the labyrinth, is a symptom rarely observed in practical otology on account of its being slightly noticeable and of short duration.

8. A large opening of the labyrinth, correctly done, with the revolving burr, is less liable to produce dangerous shaking and the breaking up of adhesions which keep the pus in a limited tract of the inner ear and do not allow it to extend, by way of the aqueducts, into the cranial cavity, this operation is one of little danger, and the mortality need not be expected to be above 4 per cent.

9. All labyrinthine fistulæ, which are only findings of evidence, without vertiginous symptoms, without tinnitus or deafness, should not be disregarded, as advised by Jansen and Hinsberg. It is advisable to open the fistula into the labyrinth immediately, the external opening of the fistulous tract being opened carefully without interference with the membranous labyrinth, removing the superficial granulations if they exist, to observe if the pus is deeply situated, and then awaiting the results of operation. If at the end of three or four months, when the operated cavity has epidermatized, the passage of communication between the middle and internal ear continues to discharge pus, and especially if vertigo and disturbances of equilibrium coexist, it is necessary to make a large opening into the labyrinth. If the fistulous tract no longer exists after epidermatization of the operative cavity the cure may be considered complete.

10. Pus can be present in the labyrinth without disturbances of equilibrium, the tests of von Stein being negative. In the unique case cited operation demonstrated pyolabyrinthitis. The whole labyrinth was destroyed, and in consequence there was complete deafness on the affected side.

11. It is necessary that the membranous labyrinth be not destroyed in order to have labyrinthine symptoms, so that the tests of von Stein will indicate the disorders of equilibrium. Where it is destroyed the well labyrinth will in time make up for the static functions which have been abolished, and the patient no longer exhibits labyrinthine symptoms.

12. The vagospinal can be wounded at its exit from the skull either

by a particle of bone (the vaginal process of the petrous bone) or by an instrument. In such a case there would be paralysis of the palate and of the vocal cord of the corresponding side, as happened in the third case cited. This tends to prove that the facial nerve has nothing to do with the innervation of the soft palate.

The Significance of Swelling of the Cervical Lymph Glands in Middle-ear Suppurations.—STENGER (*Archiv f. Ohrenheilkunde*, Band lxii. pp. 3 to 4) considers the presence of swollen lymph glands in the region of the ear and in the region of the sternocleidomastoid of great practical significance as an aid in determining the necessity of operative interference in cases of middle-ear suppuration. According to Poirier, the glands of importance in this connection are: (1) The mastoid lymph glands, ordinarily two in number, found in children connected by two or three lymph vessels over the place of origin of the sternocleidomastoid. They receive their afferent vessels from the tympanum and the posterior wall of the external auditory canal. Their efferent vessels lead into the upper group of lymph glands beneath the sternocleidomastoid. (2) The upper group of glands beneath the sternocleidomastoid, which extend from the tip of the mastoid process and the posterior belly of the digastric to the point where the omohyoid crosses the great neck vessels. An outer and inner portion of these glands can be distinguished.

In chronic middle-ear suppurations the swelling of any of these glands may be expected. In acute suppuration swelling of these glands is not the rule; but when the mastoid also is affected, one finds, as a rule, painfully swollen glands beneath the mastoid and behind the sternocleidomastoid. This glandular swelling is of especial diagnostic value in cases of acute middle-ear suppuration which run their course without especial symptoms, and in which the long-continued suppuration is the only fact pointing to an involvement of the mastoid cells. The author points out that the glandular swelling in this region may be due to other causes—*e. g.*, diseases of the scalp and tonsillar affections; and that this must always be borne in mind.

DERMATOLOGY.

UNDER THE CHARGE OF

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The Pathology of the Tuberculides in Childhood.—NOBL (*Dermatologische Zeitschrift*, Band xi., Heft 12), among 430 children suffering from various diseases, has seen the papulonecrotic tuberculide thirteen times, and accordingly believes this form of lesion is a by no means infrequent one. The eruption is composed of small nodules, pustulo-

ulcerative and necrotic, usually discrete, but sometimes grouped, having a preference for the extensor surfaces of the extremities, the lumbar region and the buttocks; it corresponds completely with the affection described by Barthelemy under the name *folliclis*. All the children in whom this form of eruption was observed without exception presented other evidences of scrofulotuberculosis, most commonly alterations in the lymphatic glands. In 11 of the 13 cases observed scrofulous gummata and lichen scrofulosorum accompanied the papuloulcerative lesions. Although it was not possible to demonstrate the presence of tubercle bacilli in a single case, their structure resembled that of actual tubercle. Tuberculin injections were regularly followed by elevation of temperature. The local phenomena either affected all the forms of eruption present, or were confined to types of lesion other than the necrotic nodules, so that no very definite conclusions could be drawn from the results.

The author believes that this form of eruption is due to organisms whose vitality has been so greatly weakened that when they arrive in the skin they die after giving rise to formative irritation. So small a quantity of toxins is produced by these weakened organisms that little or no reaction follows injections of tuberculin.

Acne Telangiectodes.—PICK (*Archiv f. Dermatologie und Syphilis*, Band lxxii., Heft 2) reports two cases of the affection which was first described by Kaposi under the name *acne telangiectodes*. Distributed over the scalp, face, upper portion of the chest, on the forearms, hands, and legs were numerous small nodules, part of which in the course of the disease underwent suppuration, part were absorbed. These lesions were most abundant in the face, and upon disappearing left depressed scars like those following acne varioliformis. Through the constant appearance of new lesions the course of the disease was an eminently chronic one, extending over many months. Histologically the lesions were composed of a granulation tissue containing numerous epithelioid and giant cells. Although many of the elements characteristic of tuberculous tissue were present, yet it was not difficult to distinguish it from such tissue. Although many sections were examined for tubercle bacilli none were found. The author's conclusions are as follows: Acne telangiectodes is an affection *sui generis*, and not identical with lupus follicularis disseminatus; but it is identical with the acnitis of Barthelemy, and must be distinguished from the disease known as folliclis. It presents no sort of etiological relationship to tuberculosis, and should be separated from the tuberculomata and the tuberculides. It does not take its origin in the sebaceous glands and, therefore, does not belong to acne. The marked implication of the sweat glands in the inflammatory changes may possibly have some etiological significance.

The Cutaneous Manifestations of Leukæmia and Pseudoleukæmia.—NICOLAU (*Annales de dermatologie et de syphiligraphie*, Nos. 8 and 9, 1904), in a paper of some length, reports the results of a clinical and histological study of two cases of cutaneous leukæmia. The first of these cases was a man with a lymphatic leukæmia pursuing a relatively slow course. In the face, upon the scalp, and in the temporal region were foci of nodules having a marked tendency to ulceration which were

subsequently transformed into soft, infiltrated, nodular plaques, more or less elevated above the surface, persisting indefinitely. There was likewise a diffuse infiltration of the lobule of the left ear, which, increasing, became a pedunculated tumor with no tendency to ulceration. Microscopic examination of the tumor of the ear revealed a structure composed exclusively of round cells having all the characters of lymphocytes lodged in a fine connective-tissue reticulum—that is, a true lymphoma. The author regards the cells of leukæmic tumors as true lymphocytes, having their origin in the blood.

The second case was one of generalized exfoliative erythrodermia with fine, dry scales, accompanied by moderate pruritus, occurring in a man, aged sixty years, presenting hypertrophy of the ganglia and spleen, with a relative increase in the percentage of the lymphocytes in the blood.

The autopsy and histological examination fully confirmed the clinical diagnosis of pseudoleukæmia. From the study of his own case and of similar ones reported by other observers Nicolau is of the opinion that the exfoliative dermatitis is not an accidental occurrence, but is directly dependent upon the pseudoleukæmia. He also discusses the relationship of this dermatitis to the pityriasis rubra of Hebra which it resembles clinically, and concludes that this latter disease is composed of three groups, one of which is closely related to tuberculosis, the second is intimately associated with pseudoleukæmia, while the third is related to none of the foregoing and should be considered as true idiopathic pityriasis rubra.

Joint Affections in Psoriasis.—MENZEN (*Archiv f. Dermatologie und Syphilis*, Band lxx., Heft 2), who has studied the records of the dermatological clinic at Bonn with a view of learning what relationship, if any, exists between affections of the joints and psoriasis, found that in only 5 out of 1000 cases of the latter disease observed during a period of twenty-three years was there any accompanying disease of the joints; 3 of these cases were studied most carefully, and in none was it possible to demonstrate any probable relationship between the disease of the joints and the eruption.

An Almost Complete Cure of Tuberculous Lupus by Permanganate of Potash.—HALLOPEAU and NORERO (*Annales de dermatologie et de syphiligraphie*, November, 1904), at a *séance* of the Société Française de Dermatologie et de Syphiligraphie, reported a case of extensive lupus which had been almost entirely cured by applications of solutions of permanganate of potash. The patient was a girl, aged fourteen years, who, since she was five years old, had had an ulcerating lupus of the face, which upon her entrance into the hospital occupied the lower two-thirds of the nose, the cheeks, and the anterior portion of the neck. The diseased areas were treated by the application of compresses wet with a solution of permanganate of potash, generally 1:50, sometimes 1:20. Under the influence of this medication the ulcerations gradually cicatrized. At the same time an extensive ulcerating lupus of the left leg was cicatrized by the same medication.

The Treatment of Port Wine Nævus by Radium Bromide.—HARTIGAN (*British Journal of Dermatology*, December, 1904) exhibited a case of

port wine nævus at a meeting of the Dermatological Society of London, in which treatment with radium bromide had been productive of decidedly encouraging results. The patient was a woman, aged twenty-six years, with a nævus covering the left cheek and the side of the nose. The two specimens of radium employed weighed 10 mgm. each, and were of the highest known radioactivity. Thirty-nine exposures, varying in duration from a half to one hour, were made at intervals of a week. At the time the patient was exhibited the nævus had entirely disappeared with the exception of a few small areas which had not yet been treated.

[The members of the Society before whom the case was presented expressed the opinion that the result of treatment was "distinctly encouraging." A photograph accompanies the report of the case, and if this picture shows the nævus at the close of the treatment, when the reporter states the mark had "entirely disappeared with the exception of a few small untreated areas," the result does not seem to the observer of the picture to be entirely satisfactory, as the photograph shows either part of the nævus remaining or scarring. If it be scarring, any treatment that will cause scarring in the treatment of nævi is to be deprecated.]

Thigenol.—SAALFELD (*Jour. mal. cut. et de syph.*, November, 1904) experimented with this new substance, put forward as a succedaneum to ichthyol. It is a composition of iodine and ichthyol, in which occurs 10 per cent. of sulphur in an organic state of combination. Thigenol possesses a dark-brown color and a syrupy consistence; is inodorous, almost insipid; soluble in water, alcohol, and glycerin. Applied to the skin it dries rapidly, and it does not stain the linen. It is a vaso-constrictor, is useful in inflammations, is drying, and favors resorption. It calms itching, as shown in 124 observations in eczema. In seborrhœic inflammations in solution it was found especially useful. It is a mild remedy, and in the author's experience was of no value in psoriasis. In phlebitis, bandaging with thigenol, glycerin, and water, equal parts, gave very good results.

Treatment of Impetigo.—HONCAMP, of Berlin (*Monatshefte f. prak. Derm.*, Band xxxix., No. 12), gives a clinical note on the value of bismuth chinolin rhodonate (Oudim Edinger), which is applied as a powder after removal of crusts, with a wooden spatula, as metal instruments are to be avoided on account of ready oxidation. At the moment of application a slight burning is experienced, which soon passes away. This can be avoided by mixing the drug with equal parts of starch. Following the application, yellow, loose crusts form which become brownish. A bandage or covering is not required. The impetigo lesions heal under the crusts in three or four days.

Clinical Results Concerning Atoxyl.—BIRINGER (*Jour. mal. cut. et de Syph.*, November, 1904, p. 848) experimented with this new drug at the clinic for cutaneous diseases at Bonn. The drug is a white, crystalline, inodorous, almost insipid powder, soluble in 20 per cent. of hot water. The solution takes on a yellowish tint on cooling, and bases to take up about 2 per cent. of atoxyl dissolved. The toxicity is about forty times less strong than that of the other inorganic arsenical preparations. Forty-two patients received subcutaneous injections of 20 per cent.

atoxyl, either in the cellular subcutaneous tissue of the interscapular region or in the muscular tissue of the thigh, the latter region being preferable. Infiltrations very rarely followed at the point of injections, and the pains were very slight. The average dose was a Pravaz syringeful every other day. In lichen rubra the treatment was exclusively by this method, the itching decreasing after seven or eight injections, on the average twenty-five or thirty being necessary for the cure. One of the patients had never tolerated arsenical treatment, but this was well borne. One case of flat wart was cured very rapidly; and one case of dermatitis herpetiformis was cured after twenty-four injections. The drug is very easy to manage and can be made of great service in cutaneous diseases.

Atrophy of the Entire Skin of Face Caused by Roentgen Rays.—Dr. Nobl (*Proceedings of Wiener Derm. Soc.*, February 10, 1904) reports the case of a man, aged thirty years, who was treated with rays for a chronic sycosis, fifty sittings, at a day's interval, five to ten minutes each, at 20 centimetres' distance, having been given. During all this long period there occurred no active reaction. The atrophic lesions developed late, and for two years subsequently had undergone no further change. The skin everywhere was thinned and had a vitreous look, with slight scaling and very fine fissures, the lines being similar to those of old age. The epidermis was rough, like tissue paper, beneath which the skin was red, showing vascular ectasias and numerous relics of the sycosis.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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The Bactericidal Activity of the Organism under Normal Conditions and during Infections, and Organism of the Body which Protect against Infection.—Hoke (*Zeitschrift f. Heilkunde*, 1904, Band xxv. p. 197) has investigated the action which extracts of various organs of the rabbit have upon the bactericidal action of normal rabbit serum. He finds that many of these extracts are capable of arresting the bactericidal power of rabbits' serum for anthrax, colon, and typhoid bacilli, the spirillum of cholera, and staphylococci. Liver, kidney, and lymph glands show the most marked antibactericidal power, while leukocytes and bone-marrow have little or no effect. The mechanism of this action was found further to be somewhat complicated and differed from the various bacteria used. For anthrax the arrest of bactericidal action is due to the absorption by the organ extracts of the immune body of the serum.

With typhoid and colon bacilli the extracts of all organs are capable of binding the complement of the serum, while lymph glands and kidney absorb the immune body as well as complement. Both the complement and immune body for staphylococci are taken up by all organs.

Further experiments showed that leukocytes and bone-marrow extracts from normal animals had practically no influence on the bactericidal action of the serum, but if one used the leukocytes and bone-marrow from an animal which had been inoculated with typhoid bacilli or one of the other types of bacteria and killed during the infection, these extracts showed a definite bactericidal action toward that particular organism with which the animal was inoculated. The activity of these extracts was independent of the serum. The extracts of other organs from the inoculated animals showed the same power of arresting the bactericidal power of the serum, as was seen with the organs from normal animals. On the other hand, agglutinins were not bound by liver, kidney, or bone-marrow cells. The author concludes that the bone-marrow is one of the most important organs in the body, in the protection of the organism against infection.

Trypanosomiasis Expedition to the Congo.—*Memoir XIII. of the Liverpool School of Tropical Medicine* (London, 1904, WILLIAMS and NORGATE) contains seven papers, the result of an expedition sent to the Congo by the Liverpool School of Tropical Medicine to investigate trypanosomiasis. The first two papers are clinical studies of the disease, and consist of the first and second progress reports of DUTTON, TODD, and CHRISTY. Large numbers of cases were studied, blood examinations of several hundred patients, and many experimental inoculations were done. The organism was found to be always the same as that discovered in 1901 by Dutton in Gambia. Out of 1172 patients whose blood was examined, 103 showed the organism, of which number but 57 were previously diagnosed as having the sleeping sickness. The authors divide the cases clinically into three types: (*a*) cases with no definite symptoms; (*b*) cases with few symptoms; and (*c*) fatal cases showing well-marked symptoms, the most notable being fever, lassitude, weakness, and wasting. This latter type is subdivided according to whether they show sleep symptoms or not. Deep or continuous sleep and lethargy, symptoms described as characteristic of sleeping sickness, were not features of the Congo disease as observed by the authors. No definite relation was noted between the temperature and pulse, and the appearance of the parasites in the peripheral circulation. The trypanosomes were repeatedly found in the cerebrospinal fluid, being discovered in 25 of 37 positive cases of sleeping sickness. In 1 case the organism was found in hydrocele fluid.

The clinical aspects of the disease were shown to vary greatly, from the presence of the organism in the blood without symptoms to cases showing much stupor, a very irregular intermittent fever, and great emaciation, followed by death. The duration of the disease the authors have been unable to determine definitely.

The third paper of the series consists of an account by the same authors of the Congo floor maggot, a blood-sucking dipterous larva, found in the Congo Free State, which they encountered during their work upon trypanosomiasis.

CHRISTY's study of the cerebrospinal fluid in sleeping sickness, based

upon 104 lumbar punctures, constitutes the fourth paper. The organism was found in the cerebrospinal fluid in 35 of the 54 positive sleeping sickness cases. It was more often present near the fatal termination of the disease. When present early in the disease, mania and other cerebral symptoms were usually prominent. The trypanosomes may never find their way into the spinal canal, and the commencement of fever or other symptoms is in no way correlated with the entrance of the parasite into the cerebrospinal fluid.

The fifth paper is a preliminary report by THOMAS and LINTON of many animal experiments for comparing the reactions of the trypanosomes of Uganda and Congo Free State sleeping sickness with those of trypanosoma gambiense (Dutton). They found no difference in the various strains of organisms, so that the name gambiense (Dutton) must be applied to all trypanosomes with which they worked. They were unable to produce any immunity against the infection and found that there was no transmission of immunity to the offspring.

Two cases of trypanosomiasis in Europeans, reported by DUTTON, TODD, and CHRISTY, and supplementary notes upon the tsetse flies, by AUSTEN, in which a revised synopsis of the species of glossina is given, complete the memoir.

Studies in Phagocytosis.—WRIGHT and DOUGLAS (*Lancet*, 1904, vol. ii. p. 1138) first showed that the phagocytic action of leukocytes is dependent upon the action of a substance in the blood plasma, which is necessary to render the bacteria available for phagocytosis. This substance they call "the opsonin."

HEKTOEN and REUDIGER (*Jour. of Infectious Diseases*, 1905, vol. ii. p. 128) have carried the work farther and have studied the action of temperature and of different chemical substances upon these bodies—opsonins—concerned in phagocytosis. They followed Leishman's method of bringing together fluids containing leukocytes and bacterial suspensions, and then counting in stained smears the average number of bacteria taken up by each leukocyte. The experiments of Wright and Douglas were confirmed and were shown to hold for the leukocytes of several species of animals not previously used. The action of the leukocytes of various animals was found to be unequal in their phagocytic powers, and there was a great difference in the amount of the phagocytosis called forth by the various organisms experimented with, the strain of pneumococcus used calling forth practically none. The authors consider that this property may be utilized to separate organisms.

No phagocytosis was observed when leukocytes washed in salt solution were exposed to bacteria that had not been in contact with blood serum, but after bacteria had been digested with normal serum they became "sensitized," and phagocytosis took place even after the bacteria so treated were washed and suspended in normal salt solution. The blood sera from various animals were found to sensitize a non-virulent streptococcus so that it was taken up by human leukocytes. Low temperatures (1° to 4° C.) retarded "sensitization," and the amount of phagocytosis was found to decrease as decreasing amounts of "sensitizing" serum were added.

The experiments of Hektoen and Reudiger show that the serum loses its sensitizing power when heated to between 54° and 60° C. Sensitized streptococci heated to 62° to 63° C. are not taken up by leukocytes

and cannot be resentitized. The authors believe that opsonins, like toxins and complements, possess two groups—haptophore and opsiniferous—which act according to Ehrlich's lateral-chain theory. When various salts and formalin were applied to the mixture of leukocyte-containing fluid and bacterial suspensions, phagocytosis was materially inhibited. This action was further shown to be principally due to the effect of the salts and formalin upon the "sensitizing" serum, while there was no effect produced on the phagocytosis when these chemicals were applied to the leukocytes alone. The authors consider that these chemical substances may neutralize or bind the opsonins so that they cannot act upon bacteria. They think that antiphocytic action of this nature may be an important factor in the establishment and progress of those infections where phagocytosis is an important factor in the destruction of the organisms.

Experimental Typhoid Fever.—Atlassoff (*Annales de l'Institut Pasteur*, 1904, vol. xviii. p. 701) gives an account of his work upon the experimental production of typhoid fever. He first turned his attention to finding some organism which, growing symbiotically with B. typhosus, favored the growth of the latter. This was done by growing typhoid bacilli upon acid media with a variety of other organisms. It was found that the typhoid bacilli would grow on a medium containing a higher percentage of hydrochloric acid if cultivated with torula rosea, than with any other of a number of organisms experimented with, and that this organism had a decidedly advantageous effect upon the growth of B. typhosus. The growth of T. rosea did not lessen the acid reaction of the medium.

The experiments were conducted upon very young rabbits, from seven to fifteen days old, when the intestinal tract is nearly free from bacteria, making it less probable for the typhoid bacilli to encounter antagonistic organisms. Four strains of typhoid bacilli, varying in virulence, were used, and were administered by the mouth together with T. rosea. In a second series of control experiments typhoid bacilli alone were used.

Sixteen young rabbits were fed with the mixed culture, 15 of which died; 3 were fed with a pure culture of typhoid bacilli, 2 of which died. Of the 15 rabbits that died after a mixed feeding, all showed hyperæmia and punctate ecchymoses of the mucosa of the intestine, and swelling of Peyer's patches. In the cases dying on the seventh or eighth day after feedings, slight ulcerations of Peyer's patches were found. The typhoid bacillus was recovered from the blood in 12 cases, and less frequently from other organs.

The author considers that he has succeeded in producing an experimental typhoid fever much like that seen in infants, where the ulcers are rarely deep. He thinks that the method of feeding gives much better promise for investigation of the disease than subcutaneous or intraperitoneal inoculation. The organism torula rosea is frequently found in the human stomach (about 70 per cent.), and may play a definite role in the etiology of typhoid fever.

Lesions of the Aorta in Syphilis.—In Germany the question has been much discussed recently as to whether syphilis produces in the aorta lesions which are typical and characteristic of that disease. A certain

type of arteriosclerosis has been described by many authors as syphilitic, and the main point at issue is the specificity of this sclerosis. CHIARI (*Cent. f. Allg. Path. u. Path. Anat.*, Band xv. p. 137, supplement) at the "VI. Tagrung der Deutchen pathologischen Gesellschaft zu Kassel" has considered in detail syphilitic disease of the aorta. As a result of an extensive research he divides arterial sclerosis in general into two types, "A" and "B." The "A" type constitutes the simple atheroma and begins in the intima. It is characterized by a swelling of the tissues, a proliferation of the local cells, fatty degeneration, and, later, regressive alterations leading eventually to the well-recognized endarteritis chronica deformans. The media and adventitia may undergo certain changes which, however, are always secondary to the disease of the intima. The "B" type of sclerosis is quite different. In this group the media and adventitia, instead of the intima, are the primary seats of disease. The surface of the aorta, macroscopically, presents marked irregularities, with corrugations, crevices, clefts, and pittings. The intima is thickened, but there is no calcification; and, further, it is to be distinguished from the "A" type by its localization in the ascending aorta and arch, the abdominal aorta being practically immune. Microscopically the media is found to be the main seat of disease. Through this coat are found small inflammatory areas, formed partly of accumulations of small round cells lying especially about the vessels, which are increased in numbers, partly by granulation tissue or fibrillated connective tissue. Giant cells of the Langhans type may be present or areas of necrosis may form the centre of the inflammatory foci. With the development of connective tissue there is a tendency to shrinkage, and thus are produced the macroscopic scarrings and corrugations over the surface of the aorta. The adventitia likewise presents inflammatory changes combined with a periarteritis and endarteritis proliferans of the vasa vasorum. To this type of affection the author gives the name of "Productive Mesaortitis." The study of a long series of cases of syphilis and progressive paralysis showed that the productive mesaortitis was present in more than half of the instances of undoubted syphilis, and in 47 per cent. of the cases of progressive paralysis. The author concludes that syphilis stands in direct causal relationship to this variety of arterial sclerosis, although with our present knowledge of this disease other etiological factors cannot be excluded. Nevertheless, the finding of a productive mesaortitis must make one think of syphilitic disease of the aorta.

In the same number of the *Centralblatt*, Benda (page 164) discusses the etiological relationship between syphilis and aneurysms. From an exhaustive critical review of the literature and a study of 6 cases of aneurysm he concludes that the small arteries and thoracic portion of the aorta are particularly susceptible to disease in tertiary syphilis. In the aorta the process affects especially the media and adventitia, producing in these situations a true gummatous inflammation, characterized by accumulations of leukocytes, lymphocytes, epithelioid cells, and Langhans giant cells, with destruction of the tissues of the vessel wall leading to necrosis, softening, and final scar formation. These changes produce a condition particularly favorable for the formation of aneurysms. Syphilis of the smaller and medium-sized arteries do not predispose to aneurysm, since the disease process in these vessels is obliterative.

Marchand (page 197) bases his observations on a study of 23 aneurysms. In only 3 of these cases was it certain that syphilis was present. He recognizes two principal varieties of arterial sclerosis corresponding to the "A" and "B" types of Chiari. The "B" type may, particularly in young persons, be ascribed to syphilis, but cannot be held as specific for this disease, or as a gummatous formation. In his preparation, the giant cells were interpreted as foreign-body giant cells. Further, this indurating form of sclerosis may favor the development of aneurysms but the ordinary form of sclerosis is generally associated with aneurysm.

Abramow (*Virch. Arch.*, 1904, Band clxxviii. p. 406) has found in five out of six aortas from syphilitics, which he has studied, alterations which correspond to Chiari's types of mesaortitis productiva. To decide as to the syphilitic nature of this process he examined during one year all the atheromatous aortas in the material at the "Nicolai" Stadtkrankenhaus of Rostow. From this study he concludes that syphilis in the aorta, as in other organs, may give rise to gummatous processes and ordinary cirrhotic changes, but that only the gummatous aortitis may be looked upon from an anatomical standpoint as specific. The other changes analogous to those described by Chiari cannot be termed specific and may occur in other conditions.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

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Standards of Ventilation.—In an address before the British Medical Association, DR. J. S. HALDANE (*Public Health*, October, 1904, p. 32) asserts that, contrary to text-book traditions, an excess of 35 volumes over the normal content of 3 volumes of carbon dioxide in 10,000 of air will have practically no effect *per se* on human beings. Experiments conducted with Mr. J. G. Priestly have shown that the percentage of carbon dioxide in the alveolar air in man "is kept constant in each individual with almost astounding precision," and that, "if the inspired air contains 50 volumes per 10,000, the change in the depth of breathing . . . is quite inappreciable to the person breathing." The increase in the proportion of carbon dioxide in the alveolar air "would certainly produce marked discomfort, if it were not so easily got rid of by a very slight increase in the depth of breathing." The deficiency of oxygen in the air of rooms he regards as of equal unimportance, and the same is true of the aqueous vapor, since the relative humidity, except in extremely close rooms and in warm weather, is usually lower inside than outside. The hypothetical volatile organic matters of respired air he regards as probably non-existent, and, therefore, devoid of influence. That they cannot be of a proteid nature, as some have asserted they are, hardly needs argument, since no volatile proteid matters have yet been discovered; and that they cannot be toxic is proved by the fact that injection of large amounts of the condensed aqueous vapor of respired air produces no more effect than that of

equal volumes of distilled water. He calls attention to the fact that schoolmasters who spend their working hours in rooms in which the air is very much vitiated by respiration have a much lower comparative mortality figure than lawyers, physicians, and farm laborers, and that their comparative mortality figure for phthisis and other respiratory diseases is even lower in proportion than the general death rate. But schoolmasters are associated with children, who are very little subject to phthisis, which is more a disease of middle age, and hence there is not the danger of infection which comes to those associated in crowded rooms with adult workers. Since the danger of infection by virulent bacilli is much greater in rooms occupied by spitting consumptives, a far higher standard of ventilation is required for such places.

In place of the legal limit of 9 volumes of carbon dioxide in 10,000 of air, fixed several years ago for cotton factories only, where the air needs to be humidified, a departmental committee, consisting of Haldane and Osborn, of the Home Office, recommended, in 1902, that one of 12 volumes for daytime and 20 for night, when gas is burning, be established for workshops generally, in the belief that great improvement would thereby be effected. This recommendation was vigorously objected to, on the ground that the proposed standard was much too lax and would render nugatory all attempts to secure efficient ventilation. Haldane insists, however, that it is impracticable to make no allowance for burning gas, for, although the products of combustion can doubtless be removed by systems of ventilating pipes, such are practically never used in factories and workshops, and would be extremely troublesome and costly; and he can call to mind no gas-lighted factory, however well ventilated, where a standard of 9 volumes can be maintained after dark in winter. An ordinary gas flame consumes five times as much oxygen and produces about three times as much carbon dioxide as an adult man; and hence a maximum standard of 20 volumes of carbon dioxide per 10,000 of air after dark appears to be reasonable. The air of elementary schools appears to be fully twice as bad as that of factories; and he recommends that if a 12-volume standard be enforced in factories, it be enforced also in schools, in spite of the necessary and very considerable expense involved.

[Anybody who has had considerable experience in the examination of school-room air knows that, in spite of all attempts to keep impurities down, the carbon dioxide is commonly far more abundant than Haldane's proposed standard would permit, especially in old buildings. Repeated examination by the writer of the air of a number of old schoolhouses at different seasons of the year yielded averages which more often reached 30 than 12 per 10,000.—C. H.]

Milk and Disease.—*Typhoid Fever.* An outbreak of typhoid fever among factory hands, the cause of which was traced to milk, is described by Dr. W. R. Stokes (*Journal of the American Medical Association*, February 25, 1905). The number of persons employed included 1500 girls and women and 400 men. The former were served with a light luncheon, which included a glass of milk, but the latter went out to neighboring places where beer could be procured. The outbreak was confined almost exclusively to the women, of whom there were as many as 200 absent at one time, but the manager believed that many were away from causes other than sickness. The milk supplied came from

a dairy where there was no history of typhoid, but bacteriological examination revealed the presence of 4,500,000 bacteria per cubic centimetre, and colon bacilli in $\frac{1}{1000}$ cubic centimetre samples. The well, which was not protected from surface drainage, was distant about fifty feet from the privy, the box of which was full of liquid fecal matter, and close to the place where the milk was cooled. Many flies were observed about the box, and they had free access to the milk-room. The well-water contained bacillus coli and 6000 bacteria per cubic centimetre, and the ice yielded bacillus coli in 1 c.c. samples and 5000 bacteria per cubic centimetre. The milk supply was cut off, and the outbreak came to an end.

Septic Sore Throat. During October and November, 1903, about 250 cases of septic sore throat occurred in the Station Ward of Woking and in a district adjacent thereto. They were fairly well distributed over the ward, and inquiry by DR. R. W. C. PIERCE (*Journal of State Medicine*, October, 1904, p. 595) into their cause led to the suspicion that they were due to milk from a farm which was the common source of supply to two dealers, who, it transpired, had complained to the farmer of the ropy character of his product. The twenty cows appeared to be healthy, but four were found to have garget, their milk containing much pus and an abundance of streptococci. The supply was shut off after November 14th, and the outbreak ceased. The farmer himself had had a severe sore throat about the middle of September, and his wife, two sons, and two daughters had been similarly afflicted during October. The majority of persons affected were adults, and among these there were eight deaths attributed to the infected milk. In many instances whole families were seized, and in one house twelve of the thirteen inmates were victims. In a number of houses invaded the succession of cases suggested the probability of contact infected. The lesions varied in character and extent. Many were indistinguishable from ordinary cases of follicular tonsillitis; others were suggestive of diphtheria, and others developed into quinsy. Usually there was considerable constitutional disturbance, and a marked tendency to involvement of the submaxillary and posterior cervical glands, which remained enlarged for a considerable length of time. The lesions were not always confined to the tonsils; in some cases the pharynx and larynx were involved, making deglutition exceedingly painful and difficult. One patient, with his nasopharynx covered with a sloughing membrane, was sent to the hospital and died in about a week of pyæmia, with a post-mortem temperature of 110°. His wife was confined about the same time and developed puerperal fever, and two of his children had sore throat. Numerous victims had pain and tenderness of the joints, following the throat symptoms in from three to seven days. In five instances very severe erysipelas followed the throat symptoms, and one of these patients developed a mastoid abscess and died in about ten days from the onset.

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